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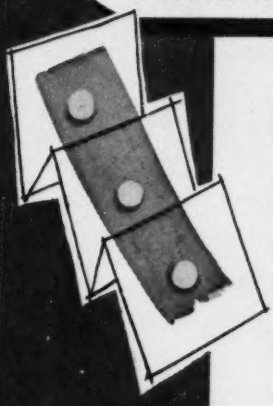

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*Keefer, C.S.: Evaluation of Antibiotic Therapy, Postgrad. Med. 9:101 (Feb.) 1951.

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J. Pediat. 39:325, 1951

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		3.8		
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	37	4.2		
	7	3.9	0.7	
	1154	4.8	2.3	8.0
	7	3.8		6.8
	27	4		
	45	3.2	3	5
	408	3.4	1.0	6.2
	184	4.5	1.3	8.3
	5	3.8		
	94	3.2	2.7	8.4
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	26	3.1	1.9	4.6
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(From Bull. of the National Research Council, No. 119, January 1950)

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1. Muller-Stephann, H.: Ztschr. Kinderheilk. 67:495, 1950.
2. Salmi, T.: Acta Paediatrica: 32:7, 1944

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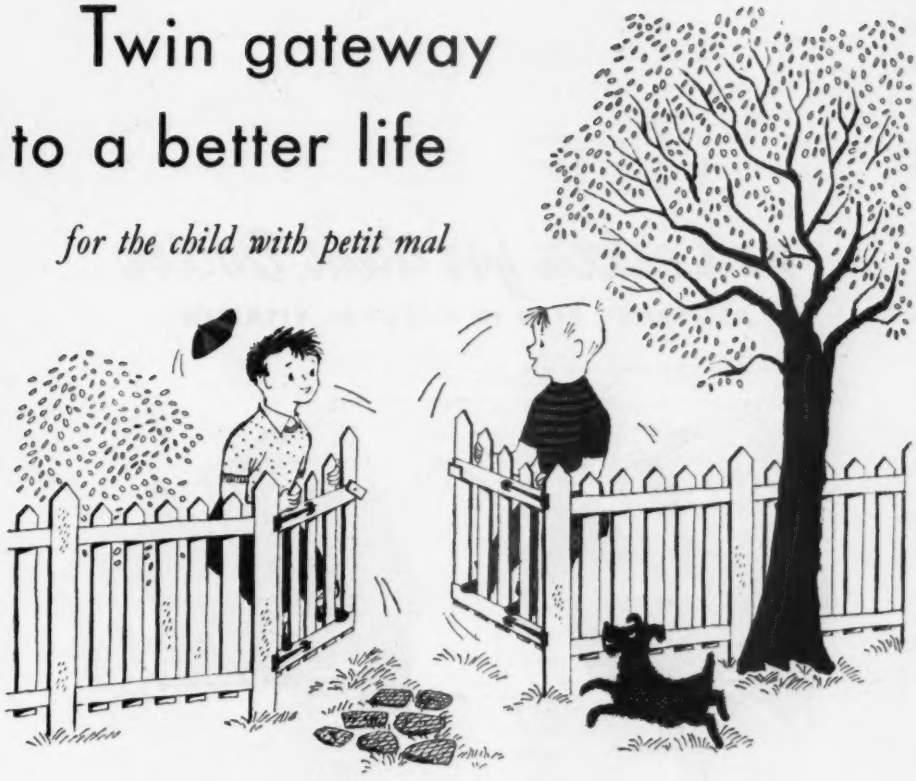

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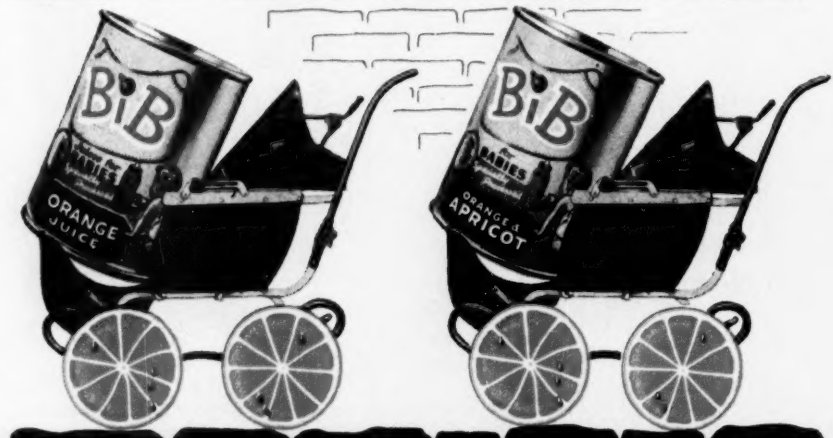

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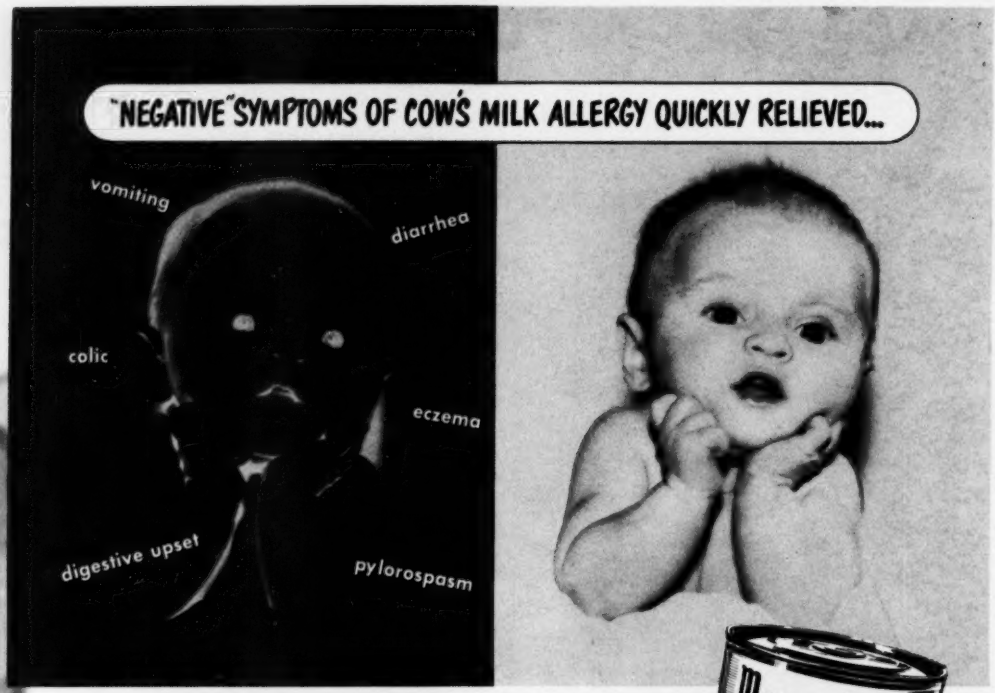

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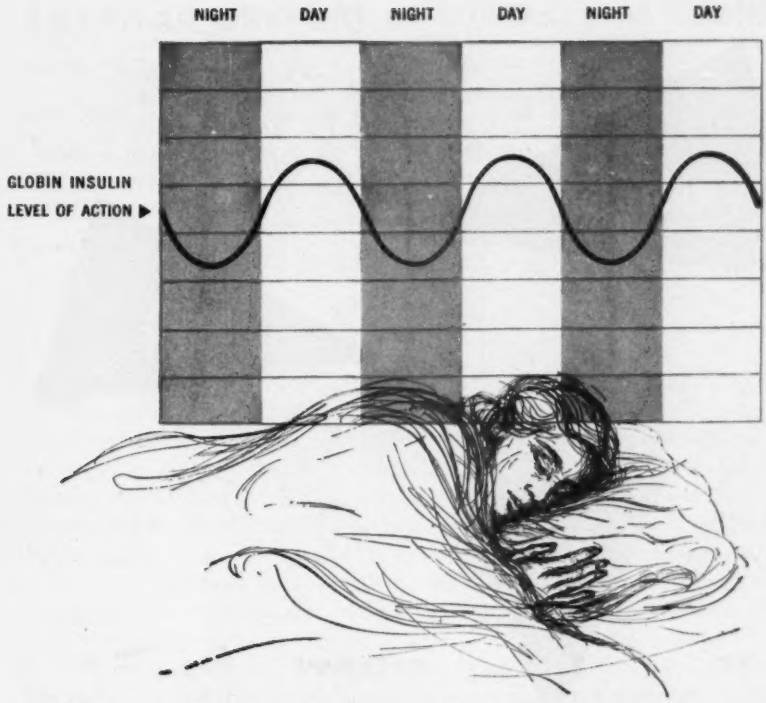

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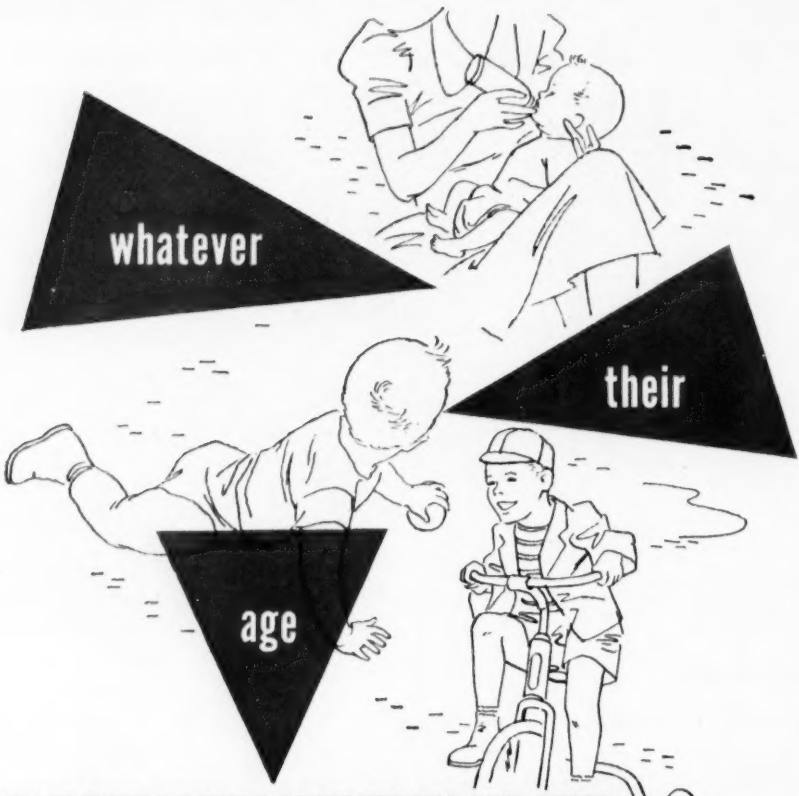

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A. M. A.
American Journal of Diseases of Children

VOLUME 83 APRIL 1952 NUMBER 4

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HERPES SIMPLEX VIRUS AS THE CAUSE OF FULMINATING VISCERAL DISEASE AND HEPATITIS IN INFANCY

Report of Eight Cases and Isolation of the Virus in One Case

WOLF W. ZUELZER, M.D.
AND
CYRIL S. STULBERG, Ph.D.
DETROIT

THE DISEASES caused by the virus of herpes simplex in man are generally thought of as benign surface infections limited to the skin and mucous membranes. In the rare instances in which the virus invades the central nervous system, the spread is believed to take place along axonal pathways.[1] Although primary herpetic stomatitis of infants is commonly associated with rather severe systemic symptoms, and the development of Kaposi's herpetiform eruption would seem to depend on hematogenous spread of the virus,[2] visceral lesions definitely attributable to the dissemination of herpes virus by the blood stream have not been recorded heretofore.[3] On morphologic grounds, however, this virus has occasionally been suspected as the causative agent for certain obscure lesions of internal organs.[4] In 1935, Hass [5] described the unique case of a premature infant showing extensive necrosis of the liver and adrenals which, because of the presence of distinctive intranuclear inclusions, he ascribed tentatively to the virus of herpes simplex. Experimentally Anderson [6] subsequently showed that at certain stages in the development of the chick embryo, the herpes virus spreads to organs distant from

---

Read before a joint meeting of the Ohio, Michigan, and Indiana branches of the Society of American Bacteriologists held in Columbus, Ohio, on Nov. 30 and Dec. 1, 1951.

From the Children's Hospital of Michigan, the Child Research Center of Michigan, and the Departments of Pediatrics and Pathology, Wayne University College of Medicine.

1. Goodpasture, E. W.: Herpetic Infection with Especial Reference to Involvement of the Nervous System, Medicine **8**:223, 1929.

2. Slavin, H. B.: The Clinical Ramifications of Infections Caused by the Virus of Herpes Simplex, M. Clin. North America **35**:563, 1951.

3. After this study was completed, a report by J. J. Quilligan Jr. and J. L. Wilson on fatal herpes simplex infection in a newborn infant appeared (J. Lab. & Clin. Med. **38**:742 [Nov.] 1951), describing the recovery of herpes simplex virus from skin and liver of a newborn infant whose mother had had herpetic stomatitis immediately preceding the birth. Though no detailed pathologic observations are given, mention is made of necrotic nodules in the infant's liver. The conclusions reached in this report are in full agreement with our own.

4. Von Glahn, W. C., and Pappenheimer, A. M.: Intranuclear Inclusions in Visceral Disease, Am. J. Path. **1**:445, 1925.

5. Hass, G. M.: Hepato-Adrenal Necrosis with Intranuclear Inclusion Bodies, Am. J. Path. **11**:127, 1935.

6. Anderson, K.: Pathogenesis of Herpes Simplex Virus Infection in Chick Embryos, Am. J. Path. **16**:137, 1940.

the site of inoculation by way of the blood stream. She demonstrated that in such embryos the virus no longer showed its customary ectodermotropism but was capable of attacking mesodermal and entodermal structures with equal ease, confirming the early observations of Goodpasture and Teague [7] on the propagation of virus directly injected into various tissues of rabbits. In Anderson's experiments the susceptibility of the embryonic vascular endothelium permitted the virus to gain entry into the blood stream and to produce metastatic lesions in many distant sites, notably the liver and spleen. The changes observed in these organs were those of massive necrosis and formation of intranuclear inclusions. Slavin and Berry [8] extended these observations to a mammalian species. Using suckling mice, they found disseminated blood-borne lesions in liver, adrenals, and bone marrow, regardless of whether the intranasal or the peritoneal route was used for inoculation.

The following observations, based on postmortem studies in eight cases, with isolation and identification of the herpes simplex virus from the liver of one infant, are presented in order to show that this virus under certain conditions produces a similar sepsis-like fatal systemic disease or hematogenous hepatitis in human infants in the absence of major cutaneous involvement.

MATERIAL

With one exception, a patient who was studied by one of us (W. W. Z.) at the Children's Memorial Hospital,[9] the patients were infants on whom autopsy was done at the Children's Hospital of Michigan between 1941 and 1951. The clinical diagnoses had varied, but the diagnosis of herpes or a herpes-like virus disease was suggested in each case by the pathologic observations, to be described. The opportunity to isolate the virus presented itself only recently in one of the cases. The identification of the virus will be described in detail. For the sake of convenience, the case material was divided into two groups, newborn infants (five cases) and infants past the newborn period (three cases).

REPORT OF CASES IN GROUP I

Case 1.—Infant L., a boy, was one of twins admitted at the age of two hours because of prematurity. The period of gestation and delivery had been uneventful. The mother, a 22-year-old primipara, subsequently stated that for years she had suffered from a recurrent vesicular, slightly pruritic eruption on the vulva. She had had two such episodes during the earlier part of pregnancy though not recently. The infant weighed 2 lb. 15 oz. (1,330 gm.) and seemed otherwise normal except for the presence of small amounts of exudate in both conjunctival sacs. It was ascertained that no silver nitrate prophylaxis had been applied to the eyes. The skin was free of eruptions. The laboratory findings at this time were within normal limits.

The patient received the usual care for premature infants, which included the injection of 1 cc. of immune globulin on admission. He seemed to do well for the first five days. At that time a purulent discharge from both eyes was noted. On the following day several small vesicles were seen on the cutaneous surfaces of both lower lids. The conjunctivae were injected and edematous. Cultures of the exudate from the left eye remained sterile. From the right eye a light growth of Staphylococcus aureus and diphtheroid organisms was obtained. In spite of irrigation of the eyes with saline and the use of aureomycin ointment, the redness and edema increased, and shortly the lids began to bleed. The skin adjacent to the right

7. Goodpasture, E. W., and Teague, O.: Experimental Production of Herpetic Lesions in Organs and Tissues of the Rabbit, J. M. Res. **44**:121, 1923.

8. Slavin, H. B., and Berry, G. P.: Studies on Herpetic Infection in Mice: III. The Visceral Lesions in Suckling Mice, J. Exper. Med. **78**:321, 1943.

9. Dr. J. A. Bigler and Dr. E. T. McEnery gave permission to publish this case.

eye became extensively excoriated. Later some of the vesicles on the lids broke, and a few similar lesions appeared in the right temporal region and on the right arm. Meanwhile, the infant's general condition deteriorated. He became listless and was in obvious respiratory distress. The chest wall was seen to retract on inspiration, and much yellow mucus accumulated in the pharynx. Blood-stained mucus and later frank blood were expectorated in rapidly increasing amounts. The temperature became unstable and on occasion rose to 102 F. The slight icterus present since the second day of life deepened. The stools became loose and somewhat frequent. In spite of therapy, which included the administration of plasma, whole blood, penicillin, aureomycin, sulfadiazine sodium, and synthetic vitamin K, the infant died on the 13th day of life.

The twin sister had done rather poorly at the start but subsequently was active, maintained a fairly stable temperature, and was not unduly icteric. At 9 days of age she became listless and had fever. Large amounts of yellow mucus were then noted in the pharynx. Cyanosis and respiratory distress developed. No cutaneous eruptions or mucous membrane lesions were observed in this infant at any time. She died at 11 days of age, two days before her twin brother. Unfortunately permission for an autopsy was not given.

Fig. 1 (Case 1).—Gross appearance of liver at autopsy, showing the numerous light necrotic nodules found.

The postmortem examination of the patient disclosed, in addition to the obvious edema and erosion of the lids and excoriation of the adjacent skin, striking gross lesions of the liver, adrenals, and lungs. These organs were riddled with pale yellow, firm necrotic nodules varying in size from 1 to 6 mm. Each lesion (Fig. 1) was surrounded by a narrow hemorrhagic zone. The remainder of the liver and much of the lung appeared dark red and hemorrhagic.

The microscopic examination showed that the pathologic process was not limited to the grossly abnormal organs but involved in addition the spleen, the kidneys, the bone marrow and the lymph nodes, the mucous membranes of the upper digestive tract, and the brain.

The microscopic picture in the parenchymatous organs was that of massive coagulation necrosis involving not only epithelial cells but also stroma and blood vessels. In the liver more than 70% of the parenchyma was estimated to be destroyed. The areas of necrosis encompassed from a few to many lobules. The walls of the portal veins, central and sublobular veins, and less commonly the branches of the hepatic arteries had undergone necrosis, as had the connective tissue of the stroma. In contrast to ordinary types of hepatic necrosis, the reticular framework and the endothelium of the sinusoids were also completely destroyed wherever the parenchymal cells were necrotic. There was virtually no cellular inflammatory response.

Hepatic epithelial cells adjacent to necrotic zones invariably contained characteristic intranuclear inclusions corresponding in every detail to the various types of inclusions amply described

in herpes simplex [10] and to those found by Hass [5] in the liver of a premature infant. They varied in appearance from large single acidophilic homogeneous or slightly granular inclusions with margination of nuclear chromatin and formation of a sharply defined "halo" to minute pink granules diffusely distributed throughout a pale vesicular nucleus. In other cells several small eosinophilic granular clusters, sometimes connected by delicate strands, were seen. Still other nuclei were completely filled by single large inclusions of more amphophilic, rarely basophilic, staining reaction which were hugged by the shrunken, distorted nuclear membrane. The cytoplasm of the involved cell often showed marked ballooning and pallor and occasionally contained minute granules similar to those seen in some of the nuclei and suggesting elementary bodies in their appearance.

In the stroma and in the endothelial cells of the blood vessels intranuclear inclusions were also seen. In such cells, as elsewhere in nonepithelial structures, the inclusions tended to be smaller yet unmistakably larger than nucleoli, from which they were also distinguishable by their eosinophilic staining reaction, the surrounding halo, and the contour, which generally tended to conform to that of the nuclear membrane.

The lesions in the adrenals were virtually identical with those in the liver, allowing only for the differences in the structure of the two organs. Here, too, massive coagulation necrosis with involvement of blood vessels and supporting stroma was evident, and again the degenerating cells adjacent to the numerous necrotic foci invariably contained many intranuclear inclusions like those already described. Only the permanent and fetal cortices were involved. No lesions or inclusions were found in the medullary parenchyma.

The pattern of the lesions was the same elsewhere. In the lungs necrosis was very extensive, involving areas covering many lobules. In the vicinity of the necrotic lesions inclusions were found in abundance. These were present chiefly in the stroma cells of the alveolar septa, the peribronchial connective tissue, the adventitia, and occasionally the intima of small blood vessels and lymphatics, in the fibroblasts of the pleura, and less commonly in the epithelial cells lining the alveoli. In studying lesions of various size and development, it was apparent that necrosis of vessel walls preceded that of the parenchyma and that the lesions spread out from the vessels in a definite radial pattern. In advance of the necrosis partially preserved cells with inclusions were always demonstrable. The bronchial lining, though sometimes detached, apparently because of necrosis of the lamina propria, was generally intact and free of inclusions. These findings suggested that the causative agent did not spread along the mucosal surfaces of the respiratory tract but reached the blood vessels first through the blood stream and extended to the surface last after producing necrosis of the intervening parenchyma.

In the kidneys a few foci of necrosis of the tubules were found, and some of the epithelial cells in such areas contained typical inclusions. In the spleen and the bone marrow small foci of complete necrosis were also present, in the latter especially near the epiphyseal lines, where the circulation is most abundant. The trabecular veins of the spleen showed a peculiar endophlebitis characterized by a spongy thickening and disruption of the intima with infiltration by red corpuscles, lymphocytes, and mononuclear cells. Rarely inclusions could be found in the wall of such vessels.

Sections of the tongue and esophagus showed a severe ulcerative process. Along the margins of the ulcers the lesions had the appearance of typical herpetic stomatitis with ballooning degeneration of the epithelium, intranuclear inclusions, and necrosis. In contrast to ordinary herpetic lesions, the necrosis penetrated deeply into the lamina propria and, in the esophagus, even into the submucosa. The deep vessels often showed marked endothelial swelling and perivascular infiltration with lymphocytes and histiocytes. Inclusions were found in endothelium, connective tissue cells, and epithelium of the esophageal glands and their ducts.

Whereas the lesions of tongue and esophagus suggested a surface type of spread, the appearance of the process in the gastric mucosa indicated that the agent reached this organ as it did the lungs, through the blood stream. There was necrosis of the small blood vessels, with

10. Scott, T. F. M.; Blank, H.; Corriell, L. C., and Crouse, H.: The Pathology and Pathogenesis of the Cutaneous Lesions of Variola, Vaccinia, Herpes Simplex, Herpes Zoster and Varicella, in Kidd, J. G., editor: The Pathogenesis and Pathology of Viral Diseases, New York, Columbia University Press, 1950.

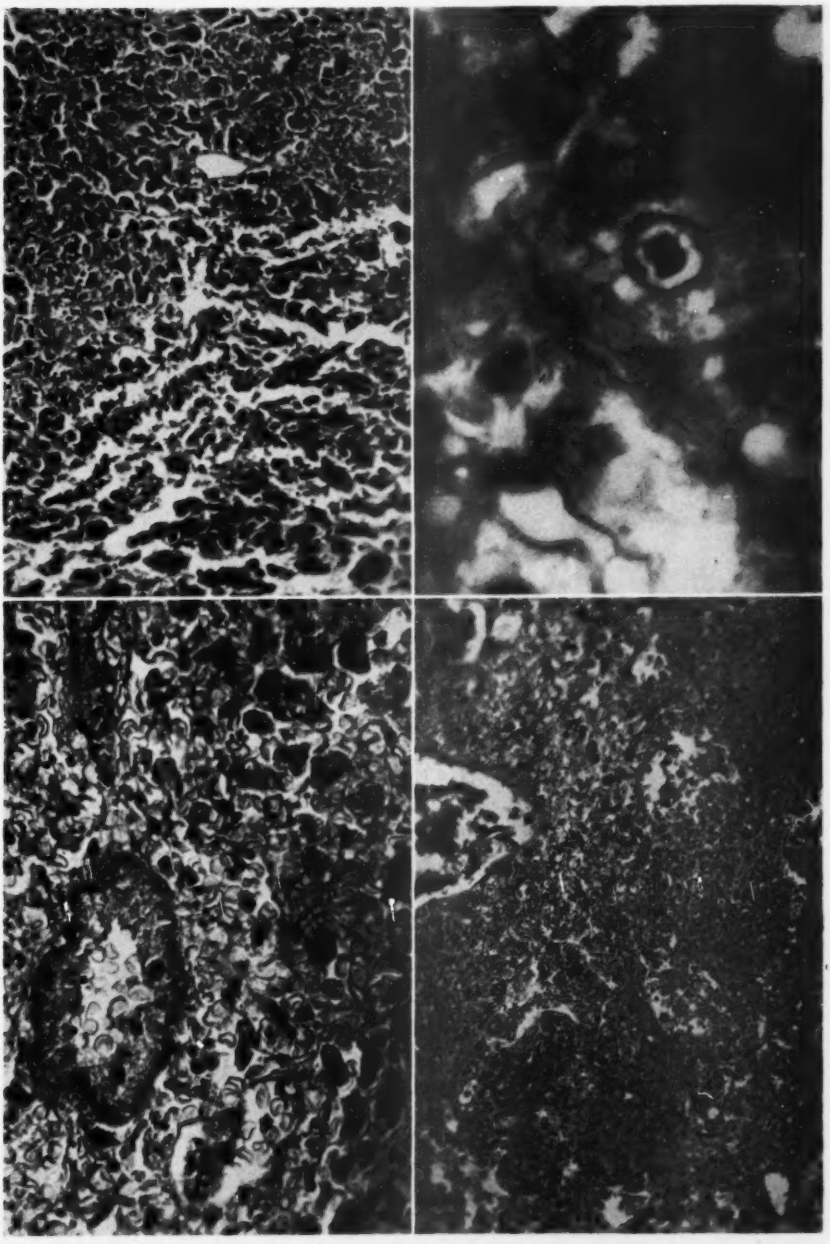

Fig. 2 (Case 1).—Upper left, margin of necrotic area in liver, showing degenerative changes and dissociation of cells in adjacent partially preserved parenchyma. Upper right, hepatic parenchymal cell, showing typical herpetic inclusion, margination of chromatin, ballooning, and granularity of cytoplasm. Lower left, necrosis of a central vein in the liver, with necrosis and hemorrhage in parenchyma. Lower right, massive necrosis of lung.

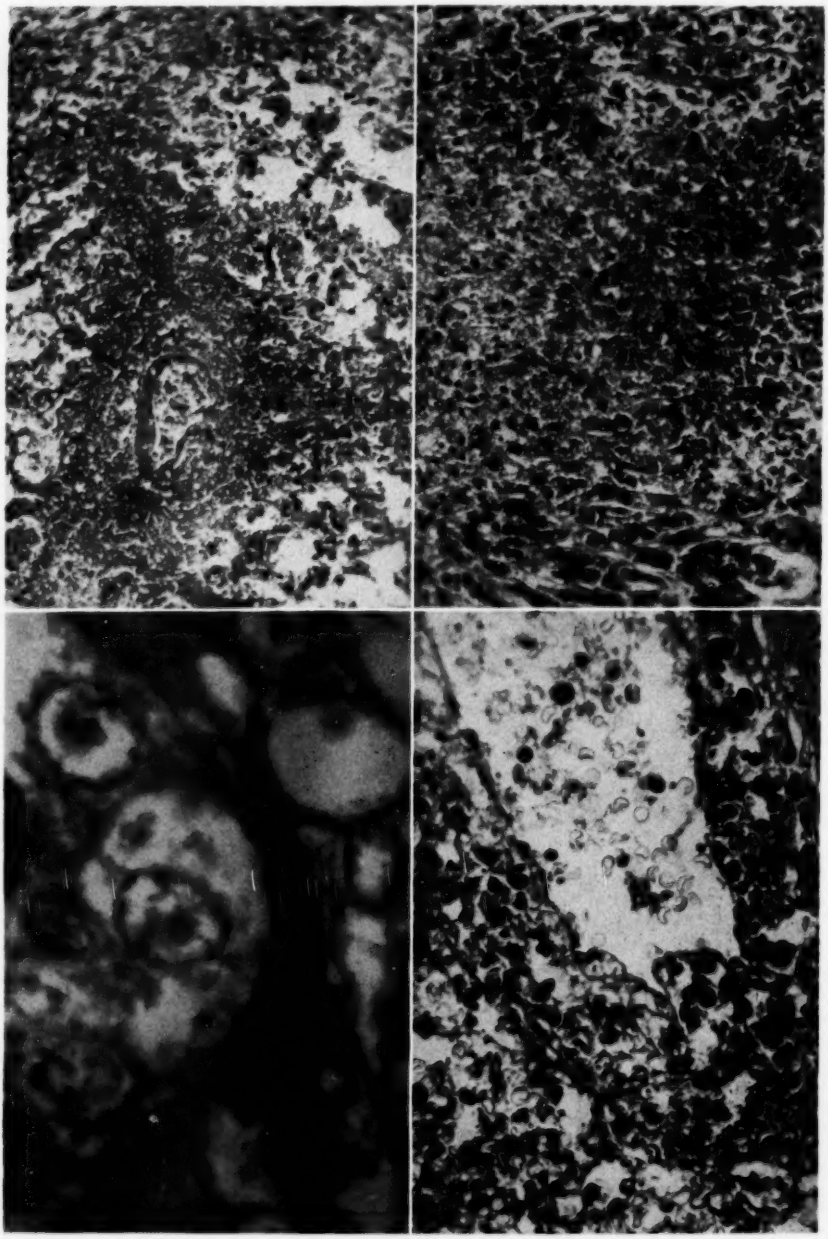

Fig. 3.—Upper left, necrosis of blood vessel and adjacent parenchyma in the lung (Case 5). Upper right, focal necrosis of adrenals (Case 2). Lower left, inclusions and ballooning degeneration of epithelium in adrenal cortex adjacent to necrotic focus (Case 4). Lower right, endophlebitis of splenic trabecular vein (Case 2).

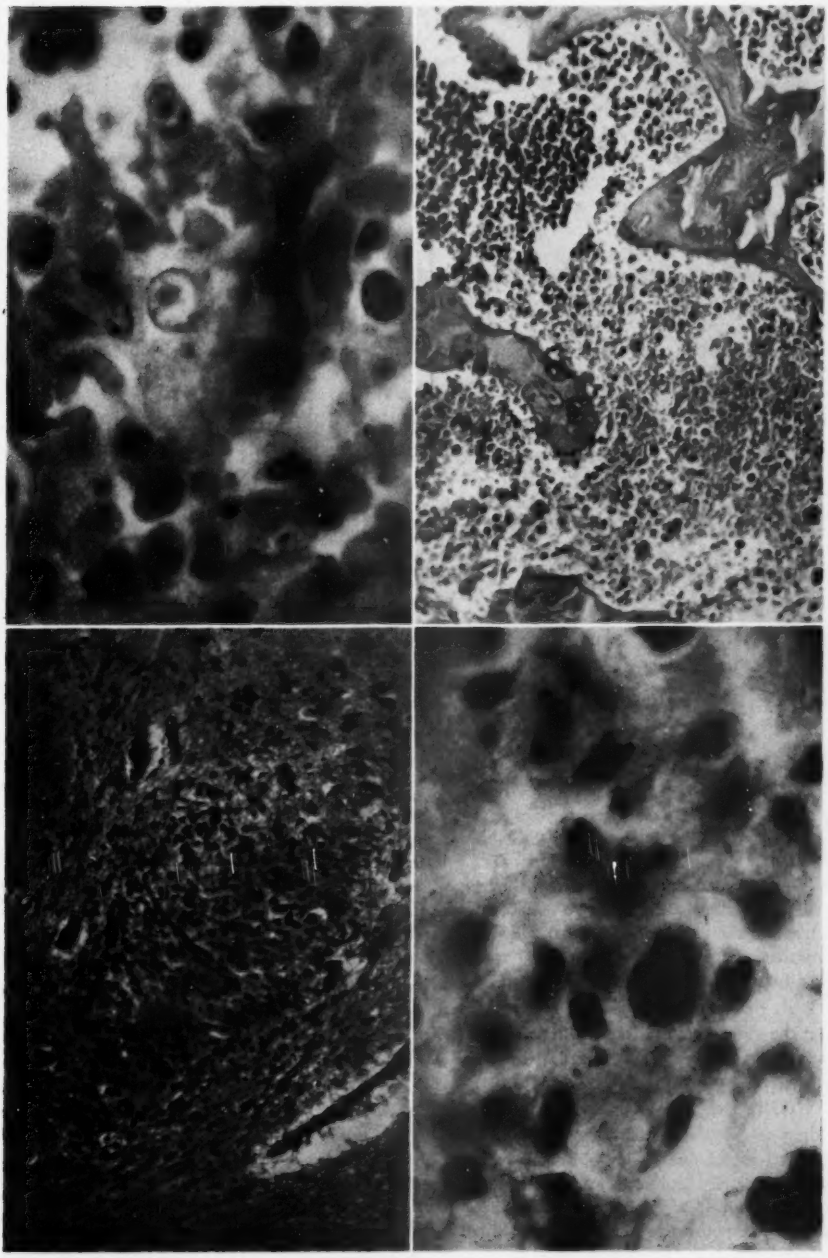

Fig. 4.—Upper left, inclusion in reticulum cell of spleen (Case 3). Upper right, necrotic lesion in bone marrow (Case 3). Lower left, localized encephalitic focus in medulla oblongata (Case 1). Lower right, inclusion in ganglion cell of medulla (Case 2).

hemorrhage and spreading necrosis of the adjacent lamina propria. Inclusions were found in the neighboring stroma but not in the relatively intact epithelial lining. No ulceration was observed.

Sections of various parts of the brain showed lesions only in the medulla oblongata. The process here was one of widespread focal encephalitis. The foci consisted of glial cells, mononuclear cells, and a few leucocytes gathered in fairly discrete nodules about degenerating ganglion cells, chiefly in the region of the olivary bodies. In such foci many cells, apparently both nerve cells and glial elements, contained large acidophilic or amphophilic intranuclear inclusions.

The skin about the umbilicus was ulcerated, and in the corium and deep subcutaneous tissue necrosis was present, but inclusions were found neither in the epidermis nor in the connective tissue beneath. Unfortunately the eyes, conjunctivae, and facial skin showing gross lesions could not be studied histologically. Nothing unusual was found in sections of salivary gland, pancreas, thyroid, thymus, pituitary, testis, lower intestinal or urinary tract, cardiac or skeletal muscle, and trachea. The pathologic diagnosis was disseminated visceral herpes simplex.

Postmortem cultures taken from the heart's blood, lungs, and liver yielded only Proteus organisms, presumably a contaminant. In smears taken from the liver a few Gram-negative rods were seen, but in sections stained with Gram's or Giemsa's solution no micro-organisms were found.

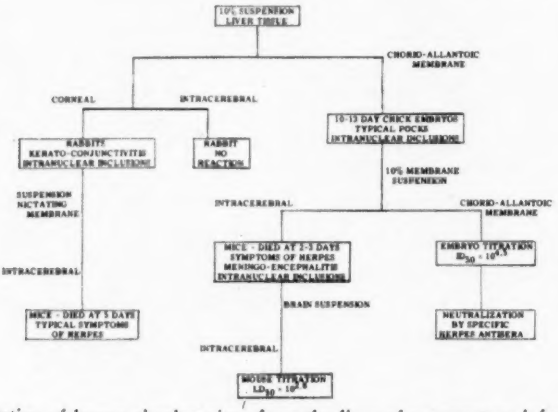

Fig. 5.—Isolation of herpes simplex virus from the liver of a premature infant (Case 1).

Isolation and Identification of the Virus.—A portion of liver was stored for 10 days at —20 C.; 0.5 gm. was removed, thawed, and ground for 15 minutes in a Potter grinder with the addition of a balanced salt solution to make a 10% suspension of tissue. Penicillin and streptomycin were added in final concentrations of 500 units and 100 γ per ml. respectively. The suspension was centrifuged at 3,000 rpm for one hour; the supernatant fluid was removed and use for inoculation (Fig. 5).

A. Primary Passage.—The corneas of four rabbits were scarified, and 0.1 ml. was inoculated on the right eye, the left serving as control. In each rabbit the inoculated eye exhibited typical keratoconjunctivitis and intranuclear inclusions of herpes simplex. The control eyes remained negative.

Of two rabbits inoculated intracerebrally, one died of the trauma and the other showed no signs of encephalitis or other infection.

A total of five 10-day-old chick embryos and ten 13-day-old chick embryos were inoculated via the chorioallantoic membrane. In all embryos plaques characteristic of herpes simplex developed within 48 to 96 hours, usually in 48 hours. Intranuclear inclusions, necrosis, and inflammatory response were evident in the three membranes examined histologically.

B. Subpassages.—Infected chorioallantoic membranes from the first generation of chick embryos served as starting material. Two subpassages through the chorioallantoic membranes of other chick embryos again resulted in typical plaque formation.

Mice: Intracerebral inoculation of undiluted membrane material (0.05 ml.) into ten 4-week-old mice resulted in death within 48 to 72 hours in all animals. On sectioning of two of the animals a meningoencephalitis with occasional inclusions in ganglion cells was seen. The meningeal reaction was characterized by necrosis of the exudate.

The brains of several mice dead from the inoculation were prepared as 10% suspensions, and their virus content was assayed by preparing decimal dilutions and injecting 0.05 ml. of each dilution into each of five other mice. As determined by the method of Reed and Muench,[11] the $L.D._{50}$ at the end of one week was $10^{-2.6}$. The virus thus showed the characteristics of recently isolated, nonadapted herpes strains in mice.

Titration of the Virus in Chick Embryos: The chorioallantoic membranes of 12-day-old chick embryos were inoculated with 0.05 ml. of progressive decimal dilutions of suspensions of membranes from the first passage. After suitable incubation, the membranes were removed and the lesions were counted. The $I.D._{50}$ (50% infectious end-point) was approximately $10^{-4.5}$. This titer is typical of recently isolated, nonadapted herpes simplex virus in the chick embryo.

Neutralization Tests: The procedure of McNair Scott[12] was followed. Table 1 shows that the virus which had undergone only one passage in the chick embryo was unequivocally neutralized by two known specific herpes simplex antisera from rabbits.[13] Serum from the patient's mother obtained 12 days post partum likewise exhibited a high concentration of specific antibody against the virus.

TABLE 1.—*Neutralization of Virus (Diluted 10^{-3}) on Chorioallantoic Membrane*

	Number of Lesions on Egg						
Serum*	Egg 1	Egg 2	Egg 3	Egg 4	Egg 5	Egg 6	Egg 7
Normal rabbit	91	>200	>200	>200	>200	>200	>200
Immune rabbit	10	11	5	12	4	3	6
Immune rabbit†	16	16	11	..	..	..	..
Human (mother of patient)	2	9	2	9	1	5	3

* A 1:16 dilution of the serum was used in each case.
† Serum was supplied through the courtesy of Dr. McNair Scott.

Comment.—The results of the animal inoculations and the neutralization tests established beyond reasonable doubt the nature of the causative agent of the infection as the virus of herpes simplex. On morphologic grounds the lesions in the various organs were regarded as characteristic of that infection even before the outcome of the virologic studies was known. The combination of pure necrosis with intranuclear inclusions of the herpes type is typical of viral lesions and would indeed be difficult to explain by bacterial action or on any other grounds, for that matter. The clinical manifestations, consisting in vesicular conjunctivitis and the appearance of vesicles and excoriations on the skin, had also pointed to the presence of the herpes virus. The history of a recurrent herpes-like eruption on the mother's genitalia is additional confirmatory evidence.

The localization of the initial lesions in the conjunctivae and on the lids together with the history of maternal infection strongly suggests that the primary infection in the infant was acquired through contact during passage through the birth canal.

11. Reed, L. I., and Muench, H.: A Simple Method of Estimating 50% Endpoints, Am. J. Hyg. **27**:493, 1938.

12. Scott, T. F. M.: Herpes Simplex in Diagnostic Procedures for Viral and Rickettsial Diseases, New York, American Public Health Association, 1948.

13. Dr. McNair Scott furnished one of the rabbit sera used in the experiment.

The additional finding of severe herpetic glossitis and esophagitis is in keeping with the assumption that infected maternal secretions also entered the oral cavity of the infant at birth.

The general distribution of the lesions is in itself evidence for the wide dissemination of the virus by the blood stream. The character of the individual lesions centering about necrotic blood vessels, with spread of the process toward the surface, further supports this concept. The uniform pathologic lesions produced by necrosis and typical inclusions in adjacent cells, and involving all tissue elements, deserve emphasis since they give rise to a pathognomonic picture on which to some extent the diagnosis of the remaining cases is based.

The illustrations of the various lesions purposely taken from several different cases will show this identity of the lesions and inclusions from case to case and from site to site (Figs. 3, 4, and 6).

The pathologic changes seen in the remaining four cases of Group I were indeed identical with those already described, varying slightly only in distribution and degree. In order to avoid repetition, these findings are given in Table 2, indicating the location and the roughly graded severity of the pathologic involvement. Because of their clinical and epidemiologic interest, the clinical histories of these patients are given in full.

TABLE 2.—*Distribution and Approximate Degree of Severity of Lesions in Cases of Group I*

Case No.	Liver	Adrenal	Spleen	Lung	Kidney	Marrow	Lymph Glands	Tongue	Esophagus	Stomach	Brain	Skin	Conjunctiva
1	3+	3+	1+	4+	1+	1+	1+	3+	4+	2+	2+	...	3+
2	4+	3+	3+	1+	1+	2+	2+	...	4+	3+	1+	1+	...
3	3+	3+	3+	2+	1+	2+	3+	...	4+	3+	1+	1+	2+
4	4+	3+	3+	...	...	...	...	...	2+	1+	...	1+	2+
5	4+	3+	3+	3+	...	2+	...	...	...	...	1+	...	...

CASE 2.—Evelyn G., an 8-day-old Negro infant, was transferred from a newborn nursery with the provisional diagnosis of sepsis and hepatitis. The infant was born by Caesarean section because of cervical stenosis in the mother and had weighed 5 lb. 10 oz. (2,550 gm.) at birth. As is customary in this nursery, she had been vaccinated with cowpox in the usual manner on the day after birth. She had done well the first four days. Thereafter, temperature ranging from 101 to 102 F. developed, and the child became listless and icteric and nursed poorly. Penicillin and sulfadiazine sodium were given, without beneficial effect. On the day of transfer periods of cyanosis were observed, and the infant regurgitated her feedings. Chest roentgenograms had shown no abnormality. The liver was significantly enlarged.

No information concerning the mother was available. On admission, apart from cyanosis, mild abdominal distention, and hepatomegaly, nothing unusual was noted. In addition to a cowpox pustule over the left arm, two small pustular lesions were seen on the trunk.

The white blood cell count was 5,200. Routine cultures of the blood and the contents of one of the pustules on the trunk were sterile.

The previous therapy was continued, and the patient also received injections of streptomycin. On the third day in the hospital icterus had become more marked, the liver had become larger, and the tip of the spleen was palpable. The prothrombin time was then over 150 seconds, the serum bilirubin concentration was 5.55 mg. per 100 cc., the total serum protein 4.9 gm. per 100 cc.; the cephalin flocculation reaction was positive (2+). It was noted that the infant bled profusely from any punctures and began to regurgitate bloody mucus. She died on the 10th day of life, 48 hours after admission.

Gross findings of the postmortem examination were almost identical with those in the first patient. Microscopically the liver was almost entirely necrotic. The spleen and bone marrow

were severely involved, as were the regional lymph nodes of mediastinum and peritoneum. In the lungs only a few small but characteristic lesions were present. The epithelium of the tongue was intact, but a severe necrotizing esophagitis was present. A single encephalitic focus with inclusions in ganglion cells was found in the medulla oblongata.

A point of particular interest was the presence of characteristic intranuclear inclusions of the herpes type side by side and sometimes in one of the same cells as the intracytoplasmic inclusions (Guarnieri bodies) of vaccinia in the cowpox pustule (Fig. 6, upper left). Together with the observation that the vessels and stroma of the corium underlying the otherwise typical pustule showed extensive necrosis, this finding suggests that the area of vaccination may have been the portal of entry for the virus of herpes simplex.

Comment.—The postmortem diagnosis in this case, studied some time before the identification of the virus in Case 1, was "generalized herpes simplex virus septicemia," a term chosen to indicate the metastatic character of the lesions and the analogy to bacterial sepsis. With Caesarean section as the method of delivery, there was no basis for transmission from the maternal genital tract. Conjunctivitis and stomatitis were not present. The lesions in the esophagus, as those in the stomach, had the appearance of a metastatic process. The presence of two bacteriologically sterile pustules on the skin (unfortunately these were not available for microscopic study) and the presence of herpes-like inclusions in the vaccinia pustule suggest the skin as an alternative portal of entry in this case.

Case 3.—Mary Jane B., a 9-day-old premature Negro infant, was transferred from another hospital because of swelling and redness of the left eye. The mother was said to be healthy. A history specifically directed toward possible manifestations of herpes was not taken. The birth weight of the infant was 4 lb. 13 oz. (2,180 gm.). For six days she had done well. Thereafter she nursed poorly and had a peculiar cry. On the eighth day the left eye became red and swollen, and a serosanguineous discharge was present. Smears and cultures of this material were negative for gonococci. The swelling progressed in spite of topical use of sulfacetamide and injections of penicillin. On the following day the infant had diarrhea, lost weight, and had grunting respirations and poor color.

On admission she appeared acutely ill. The left cornea was steamy, the eyelids markedly edematous and the conjunctiva injected. Two discrete excoriations were seen on the margin of the lower lid. The right eye appeared normal. Two large excoriated areas over the buttocks were draining serosanguineous fluid.

The temperature was 99.2 F. The white blood cell count was 16,900 predominantly composed of lymphocytes. The patient bled profusely from all needle punctures. The prothrombin time was in excess of 60 seconds. The serum protein was 4.8 gm. per 100 cc. The cephalin flocculation reaction was positive (3+). Cultures of the blood and of exudate from the left eye were sterile.

The patient quickly lapsed into a shock-like state and died within five hours after admission.

The autopsy findings were again those of necrosis in liver, adrenals, lungs, spleen, kidneys, lymph nodes, bone marrow, esophagus, and stomach. The tongue was not involved. A minimal encephalitic focus was encountered in the pons cerebri. The skin near the anus was excoriated. Severe keratitis and conjunctivitis were grossly apparent. Unfortunately the eyes could not be studied histologically. Everywhere the intimate association of necrosis with intranuclear inclusions of the herpes type was apparent.

Comment.—This case was almost identical in its clinical manifestations with Case 1, except that a history relating to maternal herpes was not available. The severe keratoconjunctivitis which was the initial manifestation of the infection suggests, especially in the light of the maternal history of the first case, a similar mode of infection during birth.

Case 4.—Phyllis G. was a 5-day-old white premature infant. During forceps delivery the physician was said to have broken the skin of the dorsum of the left hand. The birth weight was 5 lb. 4 oz. (2,380 gm.). The child had seemed well until the sixth day, when the parents were told she was sick and required hospitalization.

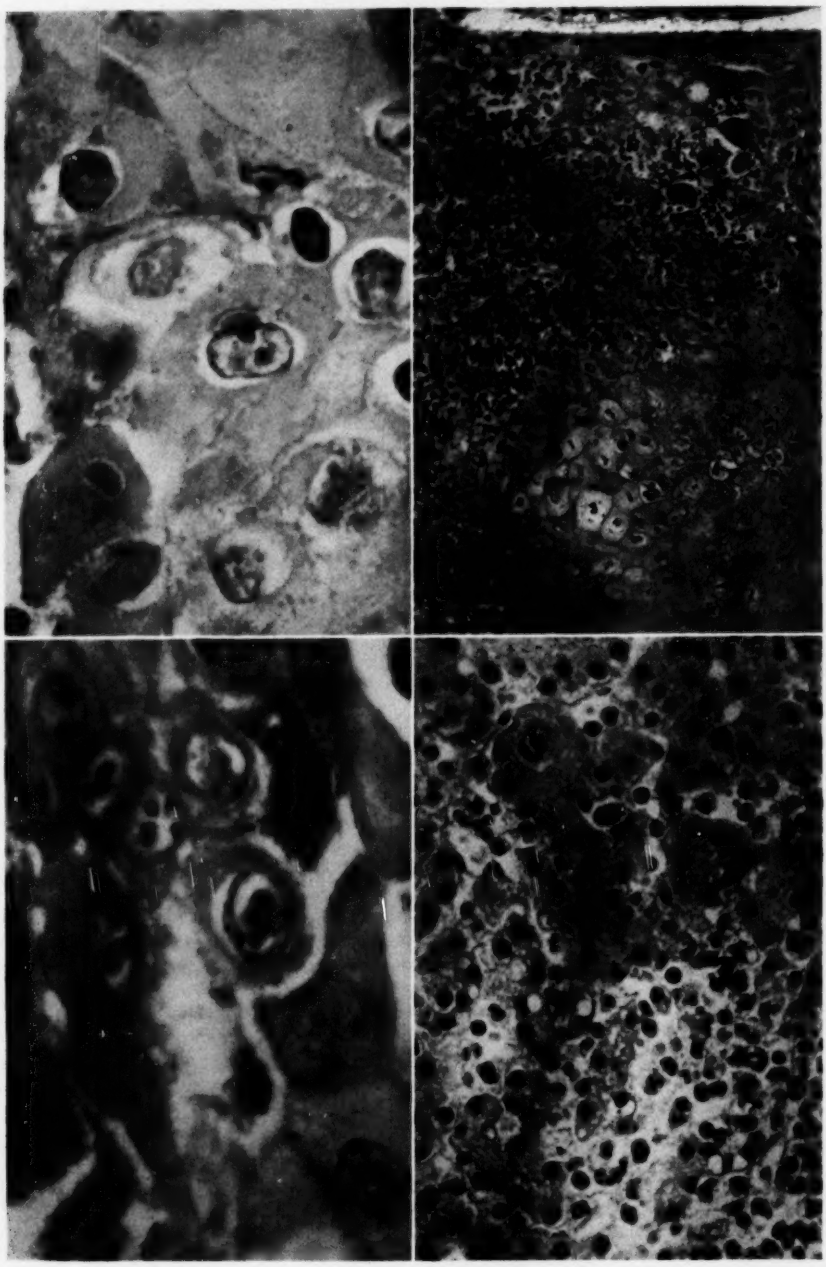

Fig. 6.—Upper left, epithelium at base of cowpox pustule, with simultaneous presence of Guarnieri bodies (cytoplasmic inclusions) and intranuclear inclusions (Case 2). Upper right, herpetic glossitis with typical changes in epithelium (Case 7). Lower left, typical inclusion at edge of esophageal lesion (Case 7). Lower right, healing focus of necrosis in liver, with inclusion in large cell at upper left (Case 8).

The infant was acutely ill. Skin and scleras were injected. The conjunctivae were reddened, edematous, and bleeding. A large ulcerated area on the dorsum of the left hand was covered with a dark brown dry scab. The temperature was 99.4 F. The infant had lost 1 lb. (454 gm.) since birth. She was given injections of dextrose, saline, and whole blood intravenously. Prolonged severe bleeding occurred from all punctures. The patient died nine hours after admission.

The autopsy disclosed massive necrosis of liver, adrenals, and spleen. Ulcers were present on the esophagus and in the cardia of the stomach. The characteristic histologic picture of the previous cases was present. In this instance, no involvement was demonstrable in the lungs, kidneys, bone marrow, lymph nodes, or brain.

Comment.—The clinical picture in this case was less well developed than in the previous ones, probably because of the rapidity with which death supervened. The obviously herpetic conjunctivitis is again of interest because of the suggested mode of infection. The case further illustrates the expected variability in the spread of the infection. In Hass's case,[5] which we accept as an unquestionable example of the same condition, only liver and adrenals were involved, and he consequently named it hepatoadrenal necrosis. Since this term describes only an accidental constellation of lesions, the term herpes virus septicemia seems preferable to us.

CASE 5.—Florence R., the only full-term infant in this group, was born two days after the mother had manifested an unexplained fever which persisted for several days after delivery. The mother, an intelligent woman of high economic standing, had not been aware of any vesicular eruption on her genitalia at any time but had experienced recurrent vulvar pruritus during the earlier part of her pregnancy. Delivery had been normal and spontaneous. The birth weight of the infant was 6 lb. 9 oz. (2,980 gm.). Except for a transient episode of cyanosis 12 hours after birth, the infant had seemed normal until the fifth day of life, when she suddenly entered into a state of collapse accompanied with dyspnea and cyanosis. The following day she vomited repeatedly. Bleeding from the rectum and vagina and severe bleeding from puncture marks were noted.

At the time of admission, at the age of 7 days, the patient was critically ill. The temperature was 95 F. The respirations were rapid and shallow. Blood was oozing from several puncture marks. Skin and scleras were icteric; the abdomen was distended; the liver was enlarged. The umbilical wound was slightly indurated. The infant was in opisthotonus position.

The white blood cell count was 17,000. The blood culture remained sterile. A chest roentgenogram showed diffuse mottling of both lung fields.

There was no response to the usual measures directed against bacterial infection, shock, anoxia, and hypothermia. The temperature fell to 94 F.; the respirations remained labored, and bloody sputum and frank bleeding from mouth and nostrils were observed. The patient died 12 hours after admission.

At postmortem examination the findings were the same as in previous cases, with maximal involvement of liver, adrenals, lungs, and spleen and focal lesions in the bone marrow. The changes in the brain were somewhat different from those in Cases 1 to 3. The lesions here consisted in early necrosis of small blood vessels, accompanied with endothelial swelling and perivascular hemorrhage. This process was limited essentially to the white matter of the cerebral hemispheres. In a few perivascular glial cells inclusions were found. No cellular infiltrations were present.

Comment.—In this case the patient was a full-term infant. The uniformity of the previous cases might have suggested that the pattern of the infection is peculiar to the premature infant, but this is obviously not the case. The systemic symptoms and the postmortem findings in this patient duplicated those in the premature infants.

This case is also the only one in the group in which no portal of entry suggests itself. There were no herpetic lesions of the eyes, oral cavity, upper intestinal tract, skin, or umbilicus. The febrile illness of the mother, beginning two days

before delivery, raises the interesting speculations of whether this infection might have been primary herpes, and whether in this instance the infection of the infant was transplacental in origin. Obviously the answer to this question cannot be given at this time. It is well to recall, however, that the earlier workers in this field claimed to have demonstrated the virus of herpes simplex in the blood of patients with active infections [14] and that, in the infants of our study at least, the presence of herpes virus in the blood is established beyond doubt.

REPORT OF CASES IN GROUP II

This group consists of three infants past the newborn period in whom at autopsy characteristic inclusions and foci of necrosis were found in the liver. Two of these patients had exhibited clinical evidence of typical herpetic stomatitis, while the third one, a debilitated infant suffering from megacolon with recurrent intestinal obstruction, had had a short febrile episode accompanied with stomatitis which was regarded as thrush and treated locally with methylrosaniline chloride.

CASE 6.—Richard C. was a 22-month-old white infant whose illness had begun with coryza. Shortly he refused his feedings and was found to have an aphthous stomatitis, for which he was treated with an arsenic preparation. The illness was accompanied with fever and listlessness and soon with complete refusal of all fluids, severe vomiting, and bleeding from the gums. The infant had been healthy and well nourished in the past.

Two weeks after the onset the condition had become critical, and the infant was admitted to the Children's Memorial Hospital. He appeared quite "toxic," acutely ill, and feverish. The gums were swollen, red, and covered with necrotic debris and old blood clots. There was a fetid odor from the mouth.

The urine contained albumin (1+) and traces of acetone. The white blood cell count was 19,800. In the differential count polymorphonuclear leucocytes predominated. Smears of the oral exudate revealed moderate numbers of fusiform bacilli and Borrelia vincentii.

Under treatment, consisting in a blood transfusion, two injections of 0.2 mg. of sulfarsphenamine, fluids parenterally, and sulfanilamide, there was no improvement. The child was alternately lethargic and restless, finally became comatose, and had repeated generalized convulsions. The temperature reached 105 F. The infant vomited large amounts of green and later bloody material, and later blood was gushing from mouth and nose almost continuously. Slight icterus developed. The patient died three day after admission in a shock-like state.

At autopsy the gross findings were limited to the marked swelling and extensive ulceration of the gingival tissues, glossitis, ulcers in the esophagus and stomach, enlargement of liver, spleen and kidneys, fatty changes in the myocardium, and small foci of pulmonary hemorrhage. The liver was strikingly pale, but no circumscribed gross lesions were present.

The histologic examination showed characteristic intranuclear inclusions in the epithelium of the tongue and in the epithelial lining of the stomach adjacent to the crater-shaped ulcers.

The sections of liver were characterized by a diffuse patchy necrosis of small groups of parenchymal cells and the presence of numerous characteristic intranuclear acidophilic inclusions, as well as margination of chromatin and innumerable mitotic figures in the surviving cells. The blood vessels, reticulum, and stroma of the portal triads remained intact. Inflammatory cells were absent.

In the kidneys necrosis of tubular epithelium and intranuclear inclusions were common. The spleen was the seat of an extensive focal necrosis, especially in the vicinity of the Malpighian corpuscles. The adrenals were not remarkable. The only changes found in the brain were those of edema and pyknosis of nuclei in the ganglion cells.

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14. van Rooyen, C. E., and Rhodes, A. J.: Virus Diseases of Man, New York, Thos. Nelson & Sons, 1948.

15. Footnote deleted.

Comment.—On pathologic grounds herpes simplex was strongly considered in the etiology of this case, but at the time there was no precedent for such a diagnosis. In the light of the cases in Group I the correctness of this view seems established in retrospect. The original clinical manifestations were those of primary herpetic gingivostomatitis as described by Dodd [16] and her associates, Burnet,[17] and others and well known to every pediatrician. The subsequent course reflects the systemic spread of the virus and particularly the hepatitis found at postmortem examination. The lesions in the digestive tract were those of herpes on mucosal surfaces and contained the characteristic inclusions. The hepatic lesion is essentially comparable with that described in Group I but shows signs of healing, probably as a reflection of the patient's more advanced age and development. The second typical affect of viruses on cells in addition to necrosis, namely, stimulation of reproductive activity, was strikingly evident in the large number of mitotic figures in the hepatic cells. The identity of the inclusions with those already described was evident. The splenic necrosis also resembled the changes seen in the cases of the first group.

In view of the usually benign outcome of herpetic stomatitis in infants, the question arises why the illness was fatal in this infant. There was no history of previous illnesses, malnutrition, or other debilitating conditions, and no complicating disease other than a terminal pneumonia was demonstrable. The question cannot be answered at this time. Perhaps viremia and visceral involvement is commoner in primary infantile herpetic disease than has been generally thought. The case then would be merely an unusually severe example in which the fatal outcome permitted the pathologic demonstration of the virus which would ordinarily remain undetected. The severity of the systemic reaction in many cases of herpetic gingivostomatitis is well known and often seems out of proportion to the significance of the local lesions in the oral cavity. There seem to be no recent studies concerning the infectivity of the blood in such cases.

The above considerations apply equally to the following case, which in turn strengthens the evidence in favor of hematogenous dissemination of the herpes virus as perhaps a frequent occurrence if not the rule in primary infections.

CASE 7.—Henry P. was an 18-month-old white boy who had never eaten well, had been subject to diarrhea, and had always breathed with difficulty. Two weeks before admission coryza developed, and one week later the infant began to cough. The disease soon assumed the typical features of whooping cough. The child had repeated convulsions and fever during the last three days.

On admission he was in a state of postconvulsive stupor. The pharynx contained a great deal of mucus. Numerous vesicles were noted on the right tonsil and the adjacent soft palate. The tongue was beefy and swollen. Liver and spleen were enlarged.

The white blood cell count was 80,000, with 78% lymphocytes in the differential count. The hemoglobin was 4.8 gm. per 100 cc. The temperature rapidly rose to 106.2 F., and further convulsions were observed. While preparations for a blood transfusion were being made the infant died.

16. Dodd, K.; Johnston, L. M., and Buddingh, G. J.: Herpetic Stomatitis, J. Pediat. **12**:95, 1938. Dodd, K.; Buddingh, J., and Johnston, L.: Herpetic Stomatitis, Am. J. Dis. Child. **58**:907 (Oct.) 1939.

17. Burnet, F. M., and Williams, S. W.: Herpes Simplex: A New Point of View, M. J. Australia **1**:637, 1939.

Postmortem culture of bronchial secretions plated on Bordet-Gengou media yielded ample growth of Hemophilus pertussis.

The gross autopsy findings were not remarkable except for cerebral edema, hepatosplenomegaly, and obvious anemia. Microscopically an intense bronchitis and peribronchitis compatible with the picture of pertussis was seen in the lungs. Sections of tonsils, tongue, and esophagus showed characteristic herpetic lesions of the mucosa with ballooning of the epithelium, intranuclear inclusions, ulceration, and necrosis (Fig. 6, upper right, and lower left).

The liver contained many microscopic foci of necrosis surrounded by cells with intranuclear inclusions and resembling in every detail the larger lesions encountered in Group I. Spleen, kidneys, adrenals, and brain showed no changes which might be attributed to herpes virus.

Comment.—In view of the characteristic lesions and inclusions in the oral cavity and esophagus, there seems to be no reasonable doubt that this case is another example of primary herpetic infection and that the focal hepatitis was likewise due to the action of this virus. In this instance death was apparently unrelated to the herpes virus, which was merely complicating a case of severe pertussis. As an incidental finding the herpetic hepatitis thus becomes of especial interest, since it provides a link between the manifest fatal disease of the previous patients and the possible undetected hematogenous dissemination of the herpes virus in ordinary nonfatal cases. The last case in the group is of similar nature, although the herpetic character of the infection had not been clinically evident.

CASE 8.—Lewis W. was admitted at the age of 3 weeks because of recurrent intestinal obstruction. The clinical picture and the roentgenologic evidence, as well as the subsequent pathologic findings, were those of a severe form of megacolon with absence of the myenteric plexus in the distal colon, and the case has been reported elsewhere in this connection.[18] The clinical course under conservative management was characterized by vomiting, recurrent abdominal distention, shock-like episodes, and weight loss and at times was complicated with diarrhea. The only noteworthy feature in the context of this paper was the occurrence, six weeks prior to death, of a short febrile episode accompanied with the development of stomatitis. The whitish plaques seen on the oral mucosa were interpreted as evidence of thrush, and methylrosaniline chloride was applied, but the lesions persisted until death.

At autopsy, apart from typical megacolon and evidence of extreme wasting, no unusual gross findings were encountered. Microscopic examination unfortunately did not include sections of tongue or tonsils. The esophageal mucosa appeared intact. In the liver numerous minute foci of necrosis were scattered at random through the parenchyma. Often the necrotic cells had already been removed, and collections of mononuclear cells had formed. Nearby, frequently single hepatic cells with large, characteristic intranuclear inclusions were found (Fig. 6 lower right). The other organs were not remarkable.

Comment.—On morphologic grounds this case must be classified with the preceding one as an example of herpetic hepatitis. The clinical evidence for an oral infection with the herpes virus is suggestive but less clear-cut than in Cases 6 and 7. The extreme debility and malnutrition of this infant may well have interfered with a more outspoken systemic response. The time interval between the febrile stomatitis and death and the relatively advanced stage of the hepatic lesions indicate that the virus maintained itself in the liver for a considerable time causing little further necrosis. This interpretation would fit the widely held belief that the virus persists in the body after the initial infection without necessarily causing further reactions in the infected tissues.[19]

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18. Zuelzer, W. W., and Wilson, J. L.: Functional Intestinal Obstruction on a Congenital Neurogenic Basis in Infancy, Am. J. Dis. Child. **75**:40 (Jan.) 1948.

19. Scott, T. F. M.: Diseases Caused by the Virus of Herpes Simplex in Rivers, T. M., editor: Viral and Rickettsial Infections of Man, Philadelphia, J. B. Lippincott Company, 1948.

COMMENT

The herpes virus seems well established as the etiologic agent of the systemic disease of the patients in Group I and of the hepatitis in Group II. The pathologic observations leading to a positive diagnosis were amply supported by clinical evidence and finally confirmed by the isolation of the virus from the liver of a patient whose case was identical in all essentials with the rest of the group.

Heretofore it has been almost axiomatic that newborn infants are not susceptible to infection with the herpes virus,[20] presumably because of protection by maternal antibody. In one case the unexplained febrile illness of a mother belonging to a high-income group, and therefore possibly not previously immunized,[17] makes tenable the explanation that lack of maternal antibody permitted the virus to establish itself in the newborn infant. In the other cases this hypothesis fails, demonstrably in Case 1, in which maternal antibody against the virus recovered from the patient was present. The fact appears significant that this patient was a premature infant like the other infants in the group and the patient described by Hass.[5] The mechanism which renders premature infants susceptible to the virus in the presence of antibody is not clear. The shortened period of gestation might lessen the transfer of antibody to the fetus, but in three of our patients prematurity was not of marked degree. The antibody concentration in the blood of the infants could not be determined.

A special susceptibility of embryonic tissues seems to offer a better explanation for the severe disease of these infants. Anderson [6] demonstrated that the stage of development of chick embryos influenced the fate of experimental herpes infection. Whereas younger embryos readily exhibited generalized lesions, the spread of the virus was not demonstrable when 18- or 19-day-old embryos were used. Kilbourne and Horsfall similarly showed that newborn mice were more susceptible to the herpes virus than were older animals. Anderson was able, however, to protect young embryos by injection of serum from hyperimmunized adult fowls. In the premature infants the maternal antibody was obviously inadequate for even partial protection.

The experience in Case 1 shows that gamma globulin, at least in small doses, cannot be relied upon even though given almost immediately after birth. The problem of protection of premature infants against herpes is obviously a serious one and involves prevention of exposure. In turn this problem has obstetrical aspects, since our findings indicate exposure during passage through an infected birth canal as one mode of infection. The existence of genital herpes in the female is abundantly proved.[22] Infants born to mothers with such lesions are liable to a special risk. It is apparent, however, that neither the mode of infection nor the portal of entry need always be the same. Exposure after birth is equally likely to lead to infection, and skin and the various mucous membranes offer equally plausible routes for the virus in a susceptible individual.

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20. van Rooyen and Rhodes.[14] Scott.[19]

21. Kilbourne, E. D., and Horsfall, F. L.: Studies of Herpes Simplex Virus in Newborn Mice, J. Immunol. **67**:321, 1951.

22. Slavin, H. B., and Gavett, E.: Primary Herpetic Vulvovaginitis, Proc. Soc. Exper. Biol. & Med. **63**:343, 1946.

The clinical picture of the infection in the newborn will be determined in part by the portal of entry. Lesions of the conjunctivae and skin such as we described should arouse a high degree of suspicion. Otherwise the cases were remarkably similar, with onset of the systemic reaction about the fifth to seventh day of life, fever or hypothermia, increasing icterus, lethargy, respiratory distress, vomiting, dyspnea and cyanosis, and the rapid development of a state of circulatory collapse. Enlargement of liver and sometimes spleen complete the similarity of this picture to that of bacterial sepsis, which will always be thought of when skin lesions or mucous membrane lesions suggest a portal of entry for bacteria. Such lesions are equally well suited to the entry of the herpes virus, and familiarity with the picture should increase the number of reported cases. It should be stressed, however, that in several instances surface lesions were absent or so inconspicuous that a clinical diagnosis of herpes did not suggest itself even in retrospect.

Special symptoms of interest are the accumulations of thick yellow mucus in the pharynx, probably due to involvement of the esophagus, and the severe bleeding tendency and hypoprothrombinemia, evidently due to hepatic failure. The degree of splenomegaly, the presence of shadows in the lungs, and other variable findings will depend on the constellation of the metastatic lesions.

Little needs to be added to the description of the pathology of the generalized disease. The pathology and pathogenesis conform in every respect to the experimental disease produced by Goodpasture and Teague [7] in rabbits, Anderson [6] in chick embryos, and Slavin and Berry [8] in newborn mice. Necrosis and production of typical inclusions are the essential features of herpes virus lesions. The detailed analysis of the lesions confirms in human material the observations of Anderson and Slavin and Berry on the mode of entrance of the virus into the blood stream through susceptible endothelial cells.

The observations on herpetic hepatitis in the older infants of Group II form a corollary to the studies in the first group. It was shown that hepatic lesions may follow typical herpetic stomatitis, and the question arises whether this event is not perhaps a fairly common occurrence, even though obvious clinical manifestations pointing to involvement of the liver are usually lacking, as they were indeed in the last two cases. It seems probable to us that at least viremia is a frequent, if not regular, phenomenon in primary herpetic gingivostomatitis of infants, in view of the occurrence of systemic reactions. The problem needs reinvestigation by testing of the infectivity of the blood of infants with primary herpetic infections and by examination of the function of the liver during and after the illness.

It is clear that visceral lesions attributable to the herpes virus are much more limited in distribution and on the whole much less severe in the older infants than in the newborn infants. Since it can be assumed that specific immunity is lacking in both groups, a nonspecific maturation of the tissues, in analogy with the animal experiments mentioned above, seems to be responsible for the differences between the two groups.

SUMMARY

The virus of herpes simplex can cause a fatal disease in the newborn in the absence of significant cutaneous lesions and can invade the blood stream during herpetic gingivostomatitis in older infants, giving rise to specific herpetic hepatitis. Pathologic evidence and clinical data, confirmed in one case by isolation of the virus

from the liver, are presented to show the development of a veritable herpes virus septicemia with metastatic lesions in five newborn infants. In three older infants a specific hepatitis was demonstrated.

The clinical features of the disease in the newborn and the pathology of visceral herpes are described in detail. The findings constitute evidence for the occurrence of herpes viremia in humans to which premature infants are especially susceptible. The pathogenesis of visceral herpes in human infants is comparable in every detail with experimental infections in chick embryos and young animals.

The mode of infection is variable. Exposure may occur during passage through a birth canal infected with herpes virus. The portal of entry may be the conjunctiva, oral mucosa, or skin. In one instance the possibility of placental transmission existed. The presence of maternal antibodies is not adequate for the protection of premature infants, probably because of a special susceptibility of their tissues. The implications of our findings for the protection of newborn and premature infants are discussed.

The occurrence of specific herpetic hepatitis in older infants with fatal outcome directly attributable to the disease raises the problem of the frequency of viremia and of visceral lesions in primary herpetic infections of infancy.

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CORTICOTROPIN (ACTH) IN THE TREATMENT OF ACUTE LARYNGOTRACHEITIS

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MEDICAL management of children with acute laryngotracheitis has long been a problem. This disease, which is confined almost entirely to the childhood period, may begin insidiously and progress to a state of anoxia, or it may strike suddenly, transforming a previously well child into a critically ill patient who is fighting for his life, truly a pediatric emergency. Many treatment regimens have been tried in the past, employing the combined skills of pediatrician, otolaryngologist, and nurse. The basic elements of moist air and sedation have remained unchanged. With the advent of antibiotics it was hoped that the disease process would be greatly retarded. The fact is, however, that this condition, despite chemotherapy and antibiotics, still remains a challenging problem.

In an attempt to attack the disease from a different angle we turned to the newer hormones. Corticotropin (ACTH) was chosen because it acts rapidly, an important consideration in severe laryngotracheitis in which time is valuable, and also because of the facility with which it can be administered, another consideration in the acutely ill child. The theory behind the use of the hormone is that stimulation of adrenocorticoids by the adrenocorticotropic hormone might be effective in modifying the defense reactions of the body over a short period of time. This could presumably occur in four ways.

1. The spreading factor may be blocked. The theory that spread of infection occurs by virtue of this spreading factor seems reasonable. It is known that pathogenic bacteria contain the substance hyaluronidase.[1] The serum of patients suffering from infections caused by streptococci, tubercle bacilli, pneumococci, etc., will augment the effect of this substance.[2] That adrenocortical extracts inhibit spreading has been demonstrated experimentally by Opsahl [3] and by Winters and his associates.[4]

From the Hospital for Sick Children and the Department of Paediatrics, University of Toronto Faculty of Medicine.

1. Duran-Reynals, F.: Studies on a Certain Spreading Factor Existing in Bacteria and Its Significance for Bacterial Invasiveness, J. Exper. Med. **58**:161, 1933.

2. Selye, H.: The Physiology and Pathology of Stress, Montreal, Acta, Inc. 1950.

3. Opsahl, J. C.: Influence of Hormones from the Adrenal Cortex on the Dermal Spread of India Ink, With and Without Hyaluronidase, Yale J. Biol. & Med. **21**:255-262, 1949.

4. Winters, C. A., and Flataker, L.: Influence of Cortisone and Related Steroids upon the Spreading Effect of Hyaluronidase, Federation Proc. **9**:137, 1950.

2. Selye [5] has demonstrated that corticotropin will enhance the phagocytic ability of the cells of the reticuloendothelial system, an important element in the defense reaction of the body against infection.

3. It is well known that one of the results of infection is an increase in capillary permeability which plays an important role in the formation of inflammatory edema. It is this phase of inflammation which is so significant in laryngotracheitis in which edema of the respiratory tract is responsible for the reduction of airway. Grenell and McCawley,[6] among others, have demonstrated that an increase in capillary permeability caused by stress is largely prevented by adrenocortical extracts or corticotropin.

4. The question as to whether the edema present in laryngotracheitis may be due in part to allergic factors is largely academic. If allergy does play a part, the use of corticotropin would be justified on the basis of its effect in inhibiting the formation and increasing the breakdown of histamine.[7]

In assessing the above theoretical reasons for expected improvement, one must consider that corticotropin therapy may produce harmful effects in bacterial infection, as reported by Kass and others,[8] Michael,[9] and Selye.[10]

In the four-month period between Jan. 1 and April 30, 1951, 17 patients with acute laryngotracheitis were admitted to the Hospital for Sick Children. In 10 of the 17 patients the condition may be classified as severe, that is, improvement with standard medical treatment was not observed. Of the 10 patients with severe infections 9 eventually required tracheotomy. There were no deaths in the series.

The age distribution is interesting, although no statistical significance may be attached in such a small series. The mean age in the group classified as mild was 23 months, while that of the patients wtih severe infections was 3¼ years. In any large series mortality figures are higher in the younger age group.[11]

The bacteria encountered in the series is shown, together with the corresponding white blood cell counts, in Charts 1 and 2. Chart 1 represents the results of cultures of sputum obtained by Auger suction [12] in patients with severe diseases. Chart 2 shows the corresponding features in those with mild disease. Once again,

5. Selye, H.: Further Studies Concerning the Participation of the Adrenal Cortex in the Pathogenesis of Arthritis, Brit. M. J. **2**:1129, 1949.

6. Grenell, R. G., and McCawley, E. L.: Central Nervous System Resistance: III. The Effect of Adrenal Cortical Substances on the Central Nervous System, J. Neurosurg. **4**:508, 1947.

7. Karady, S.; Rose, B., and Brown, J. S. L.: Decrease of Histaminase in Tissues by Adrenalectomy and Its Restoration by Cortico-Adrenal Extracts, Am. J. Physiol. **130**:539, 1940. Rose, B.: Effect of Cortin and Desoxycorticosterone Acetate on the Ability of Adrenalectomized Rats to Inactivate Histamine, ibid. **127**:780, 1939.

8. Kass, E. H.; Ingbar, S. H., and Finland, M.: Effects of Adrenocorticotropic Hormone in Pneumonia: Clinical, Bacteriological and Serological Studies, Ann. Int. Med. **33**:1081, 1950.

9. Michael, M.: The Effect of Cortisone and ACTH on Bacterial Infections, South. M. J. **44**:458, 1951.

10. Selye, H.: Influence of S. T. H., ACTH, and Cortisone upon Resistant Infections, Canad. M. A. J. **64**:489, 1951.

11. Morgan, E. A., and Wishart, D. E. S.: Laryngo-Tracheo-Bronchitis, Canad. M. A. J. **56**:8, 1947.

12. Auger, W. J.: An Original Method of Obtaining Sputum from Infants and Children, Pediatrics **2**:255, 1948.

little significance can be attached to these findings in such a small series. Indeed, the over-all bacteriological findings in large series may be misleading.[13] The interesting fact that arises is that the white blood cell counts in the patients with severe laryngotracheitis were generally high, while those in the patients with mild

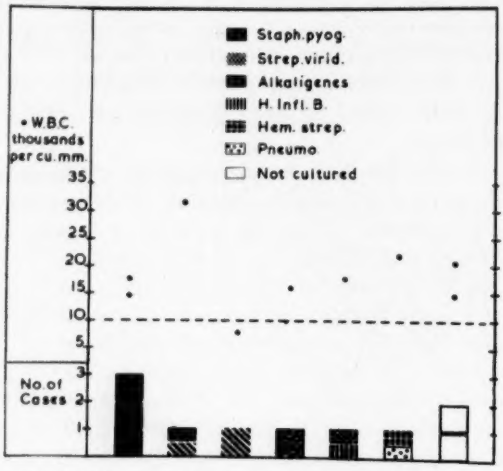

Chart 1.—Bacterial findings and white blood cell count for patients with severe laryngotracheitis.

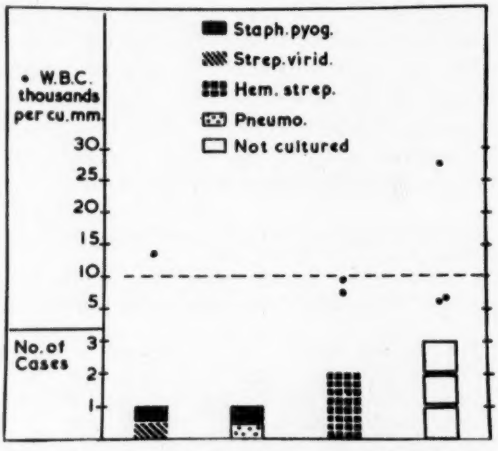

Chart 2.—Bacterial findings and white blood cell count for patients with mild laryngotracheitis.

conditions were within normal limits for the most part. All blood cultures were negative.

In the nine patients who required tracheotomy, laryngeal edema was present in six, subglottic edema in two, and diffuse edema with crusting, a distressing feature of other years, was present in only one.

13. Rabe, E. F.: Infectious Croup: 1. Etiology, Pediatrics **2**:255, 1948.

All patients with laryngotracheitis admitted to the hospital received the following routine treatment and medication: (1) Croupette Humidity and Oxygen Tent,[14] a plastic tent which delivers cooled moist oxygen, achieving an oxygen concentration at the child's head of approximately 50%; (2) sedation with secobarbital (seconal®) sodium, in a dosage, usually, of ¾ grain (0.048 gm.) rectally, repeated in one hour if necessary, and (3) penicillin and streptomycin, intramuscularly, in doses graduated according to age. Penicillin was continued in repository form until discharge. Streptomycin was administered for 48 hours only.

Four criteria for clinical improvement or deterioration were used.

Pulse. The initial pulse rate is likely to be misleading because of apprehension. The pulse rate with sedation is more significant and gives a better indication of the degree of work the heart is called upon to perform. The pulse rate is directly related to the degree of restlessness.

Respiratory rate. Again the initial rate is influenced by apprehension and is directly related to restlessness.

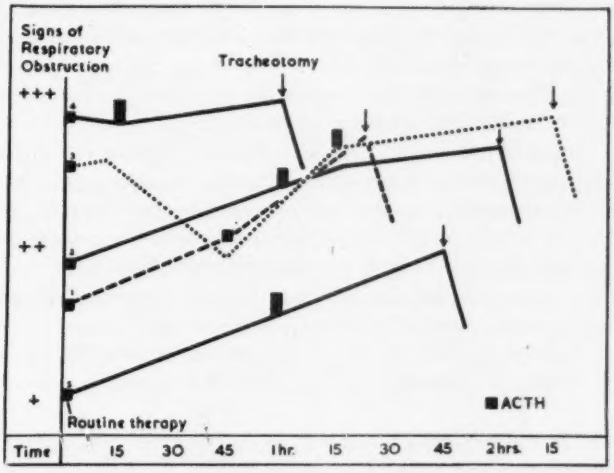

Chart 3.—Clinical course of patients with acute laryngotracheitis treated with corticotropin.

Restlessness. The restlessness associated with air hunger is a familiar sight in this disease. Increasing restlessness despite sedation and moist oxygen is of significant import, and it has been our practice to perform tracheotomy before the degree of restlessness has increased to the point of fatigue.

Air entry into the lungs and degree of indrawing. These factors are also considered in assessing the child for improvement or the reverse. Both these features are reflected in the first three signs.

Of the 10 patients with severe laryngotracheitis, 5 were selected as controls. The remaining five received corticotropin.[15] Four of the five controls required tracheotomy within two hours after admission.

The children receiving corticotropin did so following an initial observation period of not longer than two hours, during which the patients were closely

14. Manufactured by Air-Shields, Inc., Hatboro, Pa.

15. The corticotropin used in this study was supplied by National Research Council of Canada.

assessed by one of us along with a staff otolaryngologist. When improvement under the routine medical regimen was not apparent, corticotropin was administered intramuscularly followed by a further period of observation varying in length depending upon the patient's condition. The initial dose of corticotropin varied somewhat, but after the initial administration all patients received 15 mg. every four hours for a further 48-hour period.

RESULTS

Chart 3 shows in some detail the clinical course of the patients receiving corticotropin.

Case 1 was that of a 3-year-old girl who was observed for 45 minutes after the institution of routine therapy. At the end of this time, because of failure to improve clinically, 15 mg. of corticotropin was administered intramuscularly. A further period of observation was carried out for 40 minutes. During this period her condition became progressively worse, and consequently tracheotomy was performed.

Case 2 was that of an 18-month-old boy who was observed for one hour. Because of failure to improve in this time, 30 mg. of corticotropin was administered intramuscularly. During a further period of one hour, his condition steadily deteriorated, and he required tracheotomy two hours after admission.

Case 3 was that of a 4-year-old girl who was observed for one and one-fourth hours with no improvement under routine therapy. Corticotropin, 30 mg., was administered intramuscularly at the end of this time, followed by a period of observation of one and one-fourth hours. Because of failure to improve and because of restlessness and signs of fatigue, tracheotomy was performed.

In Case 4 a 12-year-old boy was admitted in marked distress. Because of this, 40 mg. of corticotropin was administered after only 15 minutes of observation. He was observed closely thereafter for a further 45 minutes during which time symptoms of obstruction became more marked, and operative intervention was required.

Case 5 was that of a 4-year-old girl who was admitted in only moderate distress, but even with sedation and humid oxygen she showed increasing restlessness, with a rise in pulse and respiratory rate during the first hour. Administration of 40 mg. of corticotropin failed to halt the progress of her difficulties over a further 45-minute period, and tracheotomy was necessary.

COMMENT

In the assessment of the results of this small series several facts arise.

1. Because only patients with severe laryngotracheitis were treated with corticotropin, we were dealing with children who were acutely and gravely ill, and, therefore, the time for observation was limited.

2. The criteria for improvement were not so objective as those in other studies in which hormone therapy has been used. Because these children were admitted, for the most part, at night and because the observation period in all cases was short, it was impossible to study such changes as eosinophile counts and 17-ketosteroid excretion in the urine. We do know from other observers [16] that a single

16. Thorn and others: The Clinical Usefulness of ACTH and Cortisone, New England J. Med. **242**:783, 1950.

dose of corticotropin will have a measurable effect in 30 minutes. Clinical improvement, however, was not noted in any of our patients during the periods of observation.

3. The dosage of drug was arbitrarily chosen. When we derived no benefit from small doses in the early patients, larger doses were subsequently given. It is possible that an increased dosage of hormone is necessary for an effective response in a short period of time, or perhaps the intravenous route may be preferable.

4. There were no appreciable differences in the results between the patients treated with corticotropin and the controls. Chart 4 shows a treated patient and a control of the same age who showed an almost-identical course, representative of the group as a whole. All were improved by tracheotomy. The postoperative course in each group was essentially the same. No evidence of overstimulation of the adrenal cortex from the use of corticotropin was noted, nor were there signs of exacerbation or spread of infection.

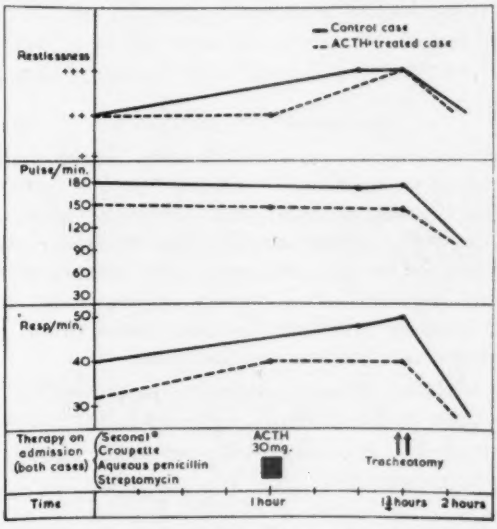

Chart 4.—Clinical course of corticotropin-treated and control patients with acute laryngotracheitis.

SUMMARY AND CONCLUSIONS

Ten patients with severe laryngotracheitis were selected for study. All received a standard medical regimen consisting of use of Croupette Tent, sedation, and antibiotic therapy. After a suitable observation period, five of the patients received an intramuscular injection of corticotropin.

No appreciable effect was noted from corticotropin administration, the clinical course in treated and control groups being identical. All but one control required tracheotomy.

The series studied is small. It is possible that larger doses or a different method of administration of corticotropin may be required. To date, however, no appreciable benefit has been derived from the use of this hormone in the treatment of acute laryngotracheitis.

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KEROSENE INTOXICATION

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IN THE area served by the Rhode Island Hospital kerosene ingestion is a frequent cause for admission to the pediatric service. Because of the debatable issues in the literature regarding its treatment, we have reviewed the cases of patients admitted to this hospital between the years 1941 and 1950, inclusive. These patients have been treated in a number of ways, and by a comparative study we shall present what would appear to be a logical method of therapy.

DESCRIPTION OF TECHNIQUE

During the above-mentioned years we have been able to study the records of 71 patients hospitalized for treatment of kerosene ingestion. Histories were examined to ascertain the following facts: time elapsed prior to hospitalization, estimated amount ingested, place obtained, and whether or not vomiting occurred. The month and year of admission and the age of the patient were also recorded. The following clinical observations were studied: symptoms and signs, classified according to involvement of respiratory, gastrointestinal, and central nervous systems; fever; genitourinary findings, and length of hospitalization.

The following laboratory data were available: white blood cell count, in 83.1% of the cases; urinalysis results, in 80.3%, and chest roentgenogram, in 62.0%.

Treatment was studied as to whether or not lavage was done. The data of the lavage study are presented in Figure 1.

RESULTS

Etiology.—Accidental ingestions occurred in the age group of 10 to 56 months, with an average of 20.3 months. A leaking stove or air vent was responsible for 23 cases. Kerosene was available in a drinking utensil on the floor in 22 cases. In the majority of the latter cases the drinking utensils were on the floor because of the leaking stove.

Seasonal Incidence.—The seasonal incidence was insignificant.

Amount Ingested.—This information was obviously difficult to obtain, and in 47 cases it was not known. The recorded amounts varied from one mouthful (5 cc.) to one cupful (240 cc.).

Clinical Observations.—These have been divided into the six following categories:

From the Department of Pediatrics, Rhode Island Hospital (Dr. Lord).

Respiratory Symptoms: All patients showed upper respiratory tract symptoms, as evidenced by coughing, gagging, or strangling. In fact, it was this symptom that attracted attention to the patient in the great majority of cases. This is in contrast to the finding of Reed and co-workers [1] that only 56.0% fell into this category. Waring [2] reported respiratory symptoms in two of 10 cases, and Steiner,[3] in 19 of 35 cases. Cyanosis was not recorded in any of our patients. Of our patients, 29, or 40.8%, were considered to have a pneumonic process. These will be discussed separately.

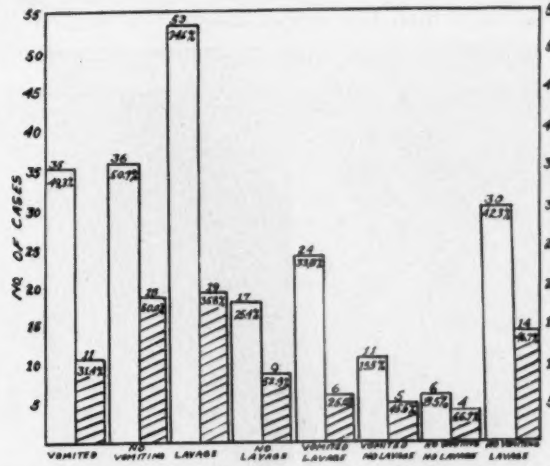

Fig. 1.—Relation of treatment to pneumonia: solid white bars indicate number of patients treated; lined bars, number of patients in whom pneumonia developed.

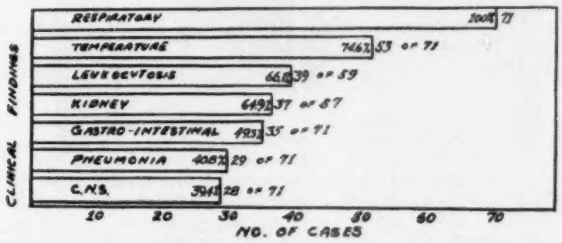

Fig. 2.—Frequency of clinical findings.

Gastrointestinal Symptoms: Abdominal complaints, as evidenced by vomiting, occurred in 35 patients, or 49.3%. No complaints of abdominal pain were recorded.

Central Nervous System Symptoms: Symptoms of lethargy, drowsiness, stupor, or convulsions occurred in 28 cases, or 39.4%. Nineteen patients were

1. Reed, E. S.; Leikin, S., and Kerman, H. D.: Kerosene Intoxication, Am. J. Dis. Child. **79**:623 (April) 1950.

2. Waring, J. I.: Pneumonia in Kerosene Poisoning, Am. J. M. Sc. **185**:325, 1933.

3. Steiner, M. M.: Syndromes of Kerosene Poisoning in Children, Am. J. Dis. Child. **74**:32 (July) 1947.

classified as slightly drowsy, seven as very drowsy or stuporous, and two patients had convulsions.

Fever: This occurred in 53, or 74.6%, of the patients, with an average of 3.5 febrile days.

Genitourinary Symptoms: These are considered under laboratory studies.

Length of Hospitalization: There was a total of 392 hospital days, or an average of 5.5 days for each patient. The average interval from ingestion of kerosene to entry into hospital was four hours, with a range of 15 minutes to two days. Forty patients were seen within one hour after ingestion and had an average hospital stay of 4.6 days. Sixty-two patients were seen in the first 24 hours, and their average hospitalization was 4.9 days. Six patients were seen after 24 hours

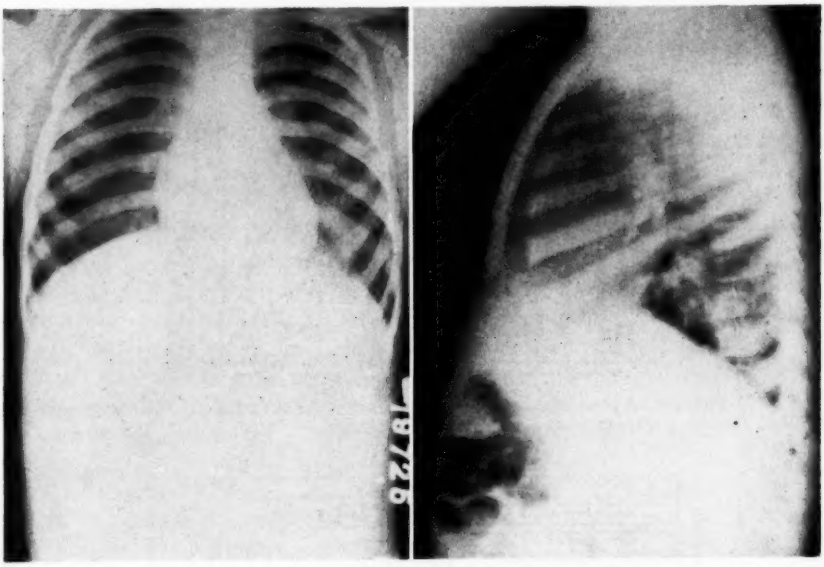

Fig. 3.—E. N., a 4-year-old white boy, ingested an unknown amount of kerosene. He was hospitalized within two hours. The findings were respiratory system involvement, no vomiting, mild central nervous system involvement, and temperature elevated one day. Treatment did not include lavage. Roentgenograms on the fourth day revealed right lower lobe pneumonia. He was hospitalized five days. Pulmonary examination showed no chest abnormalities.

and had an average of 11.3 hospital days. The interval was unknown in three cases with an average of 6.3 hospital days. Figure 1 shows the correlation of the length of hospitalization with all combinations of involved body systems.

Laboratory Data.—White blood cell counts were done for 59, or 83.1%, of the patients. They ranged from 4,800 to 29,000. The average polymorphonuclear leucocyte count was 60%. There was a leucocytosis of over 10,000 cells in 39 patients. The average white cell count for patients with pneumonia was 14,000, with an average polymorphonuclear leucocyte count of 60%. For patients without pneumonia the average white cell count was 12,100, with a range from 5,800 to 26,750; polymorphonuclear leucocyte counts for these patients averaged 55%, with a range from 26 to 81%. There was leucocytosis in 19 nonpneumonic cases.

Urinalysis was performed for 57, or 80.3%, of the patients. Thirty-seven, or 64.9%, of the patients had a positive reaction for either protein or sugar. Twenty-nine patients had proteinuria; 15 had glycosuria, and 7 had both. Test for acetone did not give a positive reaction for any of the patients. Waring [2] reported acetone in three of 10 cases.

The data presented above approximate those figures presented by Reed.[1] The frequency of clinical findings is shown in Figure 2.

Roentgenograms.—Forty-four patients had chest roentgenograms, and 26, or 59.1%, had positive roentgenographic evidence of pneumonia; 29 patients, or

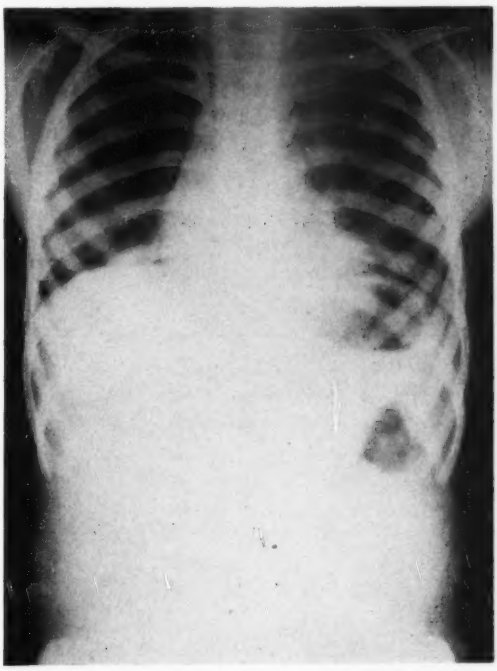

Fig. 4.—R. B., a 15-month-old white boy, ingested 1 tablespoon (15 ml.) of kerosene. He was hospitalized within one hour. The findings were involvement of the respiratory system, no vomiting, no central nervous system involvement, and temperature elevated seven days. Treatment consisted in copious lavage. Roentgenograms on the fourth day revealed bilateral lower lobe bronchopneumonia. He was hospitalized 10 days. Pulmonary examination showed no chest abnormalities.

40.8%, of the entire series were considered to have a pneumonic process. The roentgenographically demonstrable lesions in all cases were in the lower lobes. These were distributed as follows: in 5 cases in the left lower lobe, in 8 cases in the right lower lobe, and in 13 cases involving both lower lobes. This corresponds with the findings of Steiner.[3] Reed and co-workers [1] state: "The pulmonary changes were seen oftenest bilaterally and were usually most pronounced in the base of the right lung." It is interesting to note that in 19 cases with positive roentgenographic findings no positive auscultatory findings were reported. Lesser

and his associates[4] reported: "Pulmonary manifestations were observed in 77 per cent of the patients examined roentgenologically, as compared with only 24.2 per cent found by physical findings alone." The auscultatory findings described in this series of cases included coarse breath sounds, rhonchi, rales, and wheezes. Since 1946 the usual routine has included two chest roentgen examinations, one on admission and one on the fourth hospital day. After 1946 a total of 54 patients were admitted; 24 of these had pneumonia. Prior to 1946 there were 17 patients admitted, and five of these had pneumonia. For three of these pneumonia was diagnosed by roentgenography. Only four patients had chest roentgenograms prior to 1946.

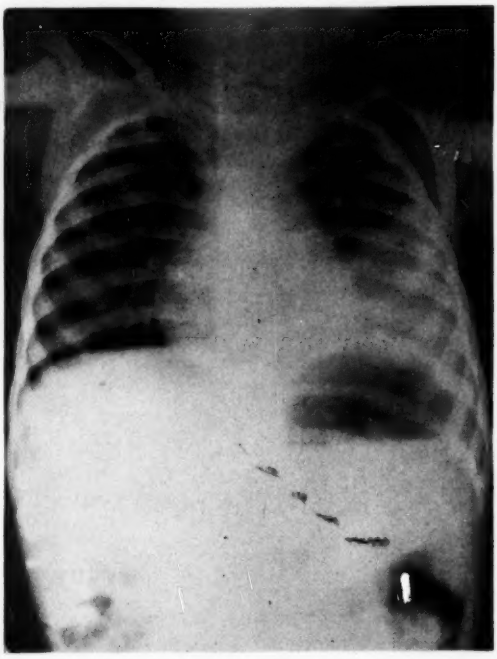

Fig. 5.—E. C., a 16-month-old white girl, ingested an unknown amount of kerosene. She was hospitalized within one hour. The findings were respiratory system involvement, no vomiting, mild central nervous system involvement, and temperature elevated 11 days. Treatment did not include lavage. Roentgenograms on the fourth day revealed left lower lobe pneumonia. She was hospitalized 19 days. Pulmonary examination showed impaired percussion note, coarse rales, and diminished breath sounds.

The patients with pneumonia had an average of 8.0 hospital days, with a range from 1 to 19 days. Representative roentgenograms and accompanying brief history and clinical course are presented in Figures 3, 4, and 5.

Treatment.—Lavage was done at admission on 74.6% of our patients, and pneumonia developed in 35.8% of these patients. Of the patients on whom lavage was not done, 52.9% had subsequent pneumonia. In 18 of 36, or 50%, of those who did not vomit and in 31.4% of those who vomited, regardless of subsequent

4. Lesser, L. I.; Weens, H. S., and McKay, S. D.: Pulmonary Manifestations Following Ingestion of Kerosene, J. Pediat. **23**:352, 1943.

treatment, pneumonia developed. Of the 24 patients receiving lavage after vomiting, in only 25.0% did pneumonia develop; in 56.7% of the patients who did not vomit and did not receive lavage pneumonia developed. This is depicted in Figure 1.

Symptomatic treatment for fever was instituted in those cases in which it was deemed necessary. Oxygen was used for one patient, and adequate sedation was given to the two patients with convulsions. No stimulants were used for any of the lethargic patients. Forty-seven patients received either antibiotic therapy or chemotherapy. Different dosage schedules were used, and the majority received two injections daily of 1 cc. of the depot type of penicillin.

There were no deaths in our series.

COMMENT

Kerosene is a hydrocarbon complex derived from petroleum. Petroleum is made up of about 50% gasoline products, 40% light fuel oil, and 10% crude oil. Until 1943 most of the kerosene was produced by distillation of the 40% light fuel oil component. In 1943 the cracking process was utilized to produce kerosene from the light fuel oil and 10% crude oil components. Kerosene has a high boiling point and a low volatility. Deichmann and associates [5] state: "Pulmonary retention of the kerosene occurs because of its low volatility, for it does not pass from the lungs readily, if at all, in the expired air." There are perhaps more impurities in kerosene obtained from the cracking process. This may be responsible for the higher incidence of pneumonia in our patients in the past five years. Kerosene, for all intents and purposes, is the same as range oil. It is used in New England as a fuel for both heating and cooking purposes in lower-income families. Years ago it was used as an expectorant in doses of 3 to 4 drops on a teaspoonful (4 to 5 cc.) of sugar.

The pathological findings resulting from kerosene ingestion reported by Deichmann and associates [5] are typical. All lobes of the lungs showed pulmonary edema and vascular congestion, with focal areas of acute interstitial inflammation and hemorrhages. The alveoli contained an inflammatory exudate consisting of fluid, fibrin, white blood cells, and foreign body giant cells. The entire tracheobronchial tree was also filled with this exudate, and the mucosa of the smaller radicles showed ulceration in places. The gastrointestinal tract mainly showed vascular congestion. The liver showed congestion of the sinusoids, slight swelling of the Kupffer cells, and fatty infiltration of the liver lobules. The principal changes in the kidneys were those of congestion, slight swelling of the epithelial cells, and precipitation of some granular material in the lumen of the convoluted tubules. The heart muscle was congested and showed some areas of muscle degeneration. In conjunction with these findings in the heart by Deichmann, the patient reported by Steiner,[3] might be mentioned; auricular fibrillation and flutter developed in this patient.

Because kerosene ingestion is a frequent emergency treated at this hospital, we undertook this study to ascertain the most logical form of therapy. Waring [2] felt that regurgitation was to be avoided and therefore questioned the use of lavage. Lesser [4] also believes that aspiration is of primary importance, and therefore he does not advocate the use of lavage unless the amount ingested was very large.

5. Deichmann, W. B.; Kitzmiller, K. V.; Witherup, S., and Johannsman, R.: Kerosene Intoxication, Ann. Int. Med. **21**:803, 1944.

Reed and co-workers [1] feel that aspiration is to be avoided and therefore lavage and the use of emetics are to be discouraged. He finds no definite relationship between lavage or vomiting and the incidence of pulmonary findings. Smith and associates [6] believe that the pneumonia is the result of both aspiration and absorption from the gastrointestinal tract. They recommend immediate and continued lavage, the use of saline cathartics, and high colonic enemas. Deichmann and associates,[5] as a result of experimental studies in animals, are convinced that absorption from the gastrointestinal tract is the primary factor producing kerosene pneumonia and the other pathological lesions. For this reason they believe all measures to remove the ingested kerosene from the gastrointestinal tract should be instituted immediately. Steiner [3] also believes that lavage should be done on these patients.

On the basis of the experience with 71 patients hospitalized at the Rhode Island Hospital, we feel that treatment should definitely consist primarily in the removal

"Combination of Symptoms" Correlated with Hospital Days

System and Temperature Involvements*	Number of Cases			Average Hospital Stay, Days		
	Without Pneumonia	With Pneumonia	Total	Over-All	Without Pneumonia	With Pneumonia
Resp.	3	1	4	4.2	4.0	5.0
Resp.; GU	2	1	3	2.0	2.5	1.0
Resp.; GI	5	..	5	2.2	2.2	0.0
Resp.; CNS	..	..	..	...	...	0.0
Resp.; temp.	3	3	6	5.7	4.3	7.0
Resp.; GU; GI	..	2	2	3.5	...	3.5
Resp.; GU; CNS	2	..	2	3.0	3.0	0.0
Resp.; GU; temp.	3	4	7	7.1	3.7	10.3
Resp.; GI; CNS	1	..	1	1.0	1.0	0.0
Resp.; GI; temp.	5	3	8	6.1	3.2	9.0
Resp.; CNS; temp.	3	4	7	6.9	4.7	8.5
Resp.; GU; GI; CNS	1	..	1	4.0	4.0	0.0
Resp.; GU; GI; temp.	5	4	9	7.3	5.4	9.8
Resp.; GU; CNS; temp.	2	5	7	7.0	6.0	7.4
Resp.; GI; CNS; temp.	3	..	3	5.3	5.3	0.0
Resp.; GU; GI; CNS; temp.	4	2	6	5.5	3.0	10.5

* Resp. indicates respiratory system; GU, genitourinary system; GI, gastrointestinal system; CNS, central nervous system; temp., temperature.

of kerosene from the gastrointestinal tract as quickly and as completely as possible. This can be most adequately accomplished with use of a stomach tube and copious amounts of water or saline. Figure 1 would seem to substantiate this form of therapy. Saline cathartics and high colonic enemas are probably also of value. It is seen that in those patients who experienced emesis significantly less pneumonia developed. The same is true of those patients on whom lavage was performed. Patients who vomited and also had received lavage had the lowest incidence of pneumonia.

There was a relationship between the length of hospitalization and the time elapsed from ingestion to admission. In general, the shorter this elapsed time, the shorter was the hospital stay. This would tend to give added support to the theory that kerosene should be removed from the gastrointestinal tract as soon as possible.

In our series of cases it was impossible to determine the exact amount ingested by each patient. However, it would seem clear that regardless of the amount of kerosene ingested the sooner lavage is performed, the better the prognosis.

6. Smith, M. H.; Waters, R. J., and Reculos, A.: Clin. Proc. Child. Hosp. 7:10, 1950.

The number of body systems involved influenced in a small way the length of hospitalization. It is seen from the table that the length of hospitalization was considerably more affected by fever and pneumonia.

The white cell count and differential smear were of no help in diagnosing pneumonia. They were approximately the same regardless of whether or not the patient had pneumonia.

In our opinion, a large number of these cases could be prevented. The largest single cause seemed to be the kitchen stove. Parents having kerosene stoves should be educated about the dangers of having leaking fuel lines and collecting drippings in a receptacle on the floor. Another common cause was the air vent for the fuel reservoir. Perhaps the manufacturers of kerosene stoves could be persuaded to locate the air vent in a less accessible place.

In a careful review of the literature we have been able to locate 325 reported cases of kerosene ingestion.[7] There are 19 deaths; the mortality rate is 5.85%. We have had no deaths in our 71 cases, and since compiling our figures have treated four additional patients without any deaths. This gives an over-all mortality of 4.75% in 400 cases. This figure certainly indicates that the problem of kerosene ingestion is not to be treated lightly.

SUMMARY AND CONCLUSIONS

Seventy-one cases of kerosene ingestion are presented and analyzed regarding varied types of treatment.

It is significant that the large majority of the cases were the result of accidental ingestion of kerosene by infants and children exposed to kerosene in easily accessible places in the family drinking utensils, which were usually used because of a defective stove. In a large number of cases the air vent for the fuel reservoir was incriminated.

A plea was made to educate families with kerosene stoves as to their potential dangers, and the suggestion was made that perhaps manufacturers could relocate the fuel reservoir air vent in a less accessible place.

The various clinical results of kerosene ingestion have been discussed, and as pneumonia was the most serious outcome, we have presented our findings as to the effect of lavage on this complication. Other manifestations were treated symptomatically.

In 19 cases, or 73.4%, with positive roentgenographic findings no positive auscultatory findings were recorded.

Treatment of kerosene ingestion should be aimed at a rapid and as thorough as possible removal of the ingested material from the gastrointestinal tract. This necessitates the use of the stomach tube and copious lavage. Although not used in this series of cases, saline cathartics and high colonic irrigations are also recommended.

There is a significant over-all mortality rate in kerosene ingestion of 4.75%.

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7. McNally, W. D., in discussion on Steiner.[3] McLean, C. C.: Kerosene Poisoning, J. A. M. A. **101**:1987 (Dec. 16) 1933. Barbour, O.: Kerosene Poisoning, ibid. **87**:488 (Aug. 14) 1926. Higgins, J. M.: Rapidly Fatal Result in a Child from Ingestion of Kerosene, Pennsylvania M. J. **36**:526, 1932. Price, J. P.: Kerosene Poisoning in Children, J. A. M. A. **99**: 214 (July 16) 1932. Nunn, J. A., and Martin, F. M.: Gasoline and Kerosene Poisoning in Children. ibid. **103**:472 (Aug. 18) 1934.

ACCELERATED REHABILITATION IN RHEUMATIC FEVER

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HERRICK House is a year-round convalescent institution for the care and rehabilitation of children recuperating from rheumatic fever. The program at Herrick House differs from that of other institutions in that mobilization is begun as soon as there is clinical and laboratory evidence that the rheumatic process is quiescent. The classic method, as opposed to this, is one of long bed rest, wheelchair, and activity-restricted convalescence.

There are a number of problems arising incident to the rehabilitation of the rheumatic patient which are different from those arising in other illnesses. There is no exact method by which the activity or quiescence of the disease may be determined. The possibility of recurrences, as in no other disease, is a sword of Damocles that threatens the physician as well as the rheumatic fever patient. The protracted period of hospitalization necessary for the treatment inevitably results in marked anxiety for the child and his family. The amount of heart damage which is present, or which may become evident over the years, carries with it the threat of disability, and not only promotes apprehension and anxiety in the patient and his family, but also places a responsibility on the physician. The physician must plan the patient's future in terms of physical activity so that it will promote maximum health in the patient.

This threefold dilemma of recurrences, anxiety, and heart disease produced the classic methods of rheumatic convalescence and rehabilitation. In many places in the country today the belief is still prevalent that the rheumatic fever patient should convalesce very slowly, remaining in bed and then in a wheelchair long after evidences of active infection have disappeared. Even when he returns to his home, his family is advised to limit his activity. He is not allowed to run and play as others do; as he grows he is restricted completely from engaging in sports and school gymnasium classes. These recommendations are frequently made on the basis of heart murmurs alone without particular regard to the state of the myocardium

This research was supported by a grant from the Chicago Heart Association.

A preliminary report of this work was presented before the First World Congress of Cardiology, held in Paris, France, Sept. 3 through 9, 1950.

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and the dynamic significance of the valvular lesion in terms of heart size and exercise tolerance. The result of this treatment often has been an activity-restricted child whose mental crippling far exceeds the severity of the cardiac defect.

Another and an antithetical course of action is one which is referred to at Herrick House as accelerated rehabilitation. There is a paucity of institutions designed to minister to youngsters convalescing from rheumatic fever. Herrick House is one of these few. It was established as a part-time program in 1922 and in 1946 was opened as a full year-round institution. It has the atmosphere of a camp, rather than a hospital—no uniforms, white or any other color, are worn. It is located in Bartlett, Ill., about 30 miles west of Chicago, on an 80-acre tract which includes a small lake and a large wooded area. The basic philosophy at Herrick House is that rheumatic children may be mobilized fairly rapidly under careful medical supervision as soon as there is clinical and laboratory evidence that the rheumatic process is quiescent, and that such mobilization is not injurious to their hearts. The philosophy was formulated and developed under the guidance of the Medical Advisory Committee [1] and three former attending physicians, Drs. Robert Herzog, George Eisenberg, and Carl Marienfeld. Children ages 6 to 15 years are accepted at Herrick House as soon as their active process is quiescent, and they come from the many large hospitals in the city of Chicago, from all over the state of Illinois, referred by the Division of Services for Crippled Children, and by referral from private physicians. This results in a variation in the medical care and status of the patients. Some of these patients are not ready for ambulation. In general, those children coming from private institutions, as compared with those from the large charity hospitals, seemingly have had longer periods of hospitalization and have fewer evidences of a residual active rheumatic process.

When they first enter Herrick House, they are brought to the infirmary. There they are kept in bed until a complete physical examination has been done and laboratory procedures, including urinalysis, blood counts, sedimentation rate determinations, electrocardiograms, and chest roentgenograms, have been completed. In some cases antistreptolysin titers are determined.

When it is our clinical impression from observation, examination, and laboratory data that the rheumatic process is quiescent, mobilization is begun. This first consists of very sedentary activity. The child is given bathroom privileges, allowed to eat at the table in the infirmary, and may watch television programs. If all goes well, the child is moved within a few days from the infirmary to one of our four dormitories, which are set up with counselors in charge, and divided according to ages, so that the children live in four groups: little girls, big girls, little boys, big boys. The counselors are young college graduates who have had training in the social sciences and whose interests are in group work. They are alert to both the physical and the emotional needs of the children.

The physical activity of the child is increased slowly, with the result that over a period varying from two to five months, depending upon the rapidity with which he reaches maximum tolerance, the child goes from bed rest with bathroom privileges (called Stage 0) to full activity (Stage 4), which may include rowing, baseball, hikes, swimming, dancing, ice skating, and sledding. Children with severer

1. Drs. Herrick, Bay, Black, Gibson, Gilbert, Grulee, Kagan, Katz, Poncher, Rosenblum, Strauss, and Volini (deceased).

heart damage and diminished cardiac reserve progress more slowly. Between Stage 0 and Stage 4 there are intermediate stages of graded activity.

There are frequent reexaminations as the child is promoted through these activity stages. In addition to physical examination and fluoroscopy, carried out at intervals of two weeks, there are sedimentation rate determinations every 10 days for the first month, then at three-week intervals thereafter. Temperatures and pulse rates are determined once a day. Sleeping pulses are determined when deemed necessary. Counselors watch for certain symptoms, such as fatigue, shortness of breath, coughing, restlessness, nosebleeds, sore throats, and running noses. These are reported immediately to the nurse and doctor. Table 1 gives a detailed list of activity stages.

TABLE 1.—*Activity List for Progressive Mobilization in Convalescence and Rehabilitation*

Stage 0 (Infirmary)
1. Bed rest
2. Bathroom privileges
3. Eat at table in infirmary
4. One activity a day in wheelchair
5. Television
6. Boat or car ride on individual basis

Stage 1 (some children may continue living in the infirmary when in Stage 1)
1. Freedom of court
2. To table (dining room)
3. To school
4. Walk to the lake
5. Sedentary or quiet group activity
6. Rides in car or boats; crafts, dramatics
7. Pushed on swings

Stage 2
1. Fishing
2. Short hikes (½ mile)
3. Car rides; boat rides
4. Horseshoes
5. Shuffleboard
6. Unlimited play on playgrounds
7. Mild swinging

Stage 3
1. Hikes (2 miles)
2. Rowing (20 minutes)
3. Baseball, no running bases
4. Archery
5. Swings (20 minutes)
6. Short volleyball games (20 minutes)
7. Out-of-camp trips
8. Folk dancing or social dancing, no jitterbugging
9. Skating and sledding (20 minutes)

Stage 4
1. Rowing (1 hour)
2. Baseball, can run bases
3. Hikes, unlimited
4. Swings
5. Swimming

During the course of rehabilitation counselors are instructed to watch for and report to doctor or nurse the following symptoms: (1) fatigue, (2) shortness of breath, (3) coughing, (4) restlessness, (5) nosebleeds, and (6) colds, sore throats, and running noses.

The child is advanced through successive stages of physical activity as his condition warrants. Severe heart damage will limit the activity of a few children. These children will reach a plateau of tolerance at the mildly restricted Stage 3 level, and sometimes even at Stage 2. Each patient is given individual attention, and activity tolerance is gauged by an estimate of the child's exercise tolerance correlated with the amount of heart damage. For many patients without evidence of residual heart damage the physician may increase activity by elevating the patient from Stage 1 to 3, or Stage 2 to 4. Thus, the program consists in graded physical activity combined with careful medical observation.

During the period of convalescence many emotional problems become evident. Some of these were present before the illness began; some were brought on by the illness. In almost all cases there is a certain fertile soil for emotional upsets because of the age of the patient, the family background, the economic status, and the environment. An attempt is made to analyze and understand the child's

emotional difficulties. This is done by direct interview and staff consultation with the psychiatrist, Dr. Irene Josselyn. Under her expert guidance the child's adjustment is improved by environmental manipulation so that the child may mature emotionally to as great an extent as possible in the short period he is at Herrick House. Frequently bizarre, aggressive, or belligerent behavior which puzzles the staff may be explained by the psychiatrist as part of beginning healthy emotional maturation. At these times it takes great patience and understanding to guide these children into the paths of healthy growth. A little straw is thrown into the emotional balance with the hope that the small beginning and readjustment of maturation will continue after the patient returns home. When considered necessary, and if it is possible to do so, arrangements are made for psychiatric follow-up, and when advisable, foster home placement is recommended. Arrangements of this type, as well as recommendations for changes in home environment, physical and human, are carried through by the case worker.

Along with the attention to the physical and emotional well-being of the child, there must be adequate interpretation of the child's illness, both to the child and to his parents. This interpretation must first explain to the parents what rheumatic fever is, what we know about it, how it acts, and what the real dangers are for the child. It must be individualized so that the parents are in a general way aware of the cardiac state of their child and of the advisability of the imposition or elimination of restrictions. Part of the interpretation to the parents is done by direct meetings with the physician, and part is done in large group meetings where the physician and social worker talk to the entire parent group. A question and answer period follows a prepared presentation. This gives the parents opportunity to recognize that their problems are not unique.

The social worker in the setting is responsible for receiving children at Herrick House and giving continued attention to those with special problems. She gives individual help to staff members working with children with special needs, and often works directly with the children themselves. This serves to implement the recommendations of the psychiatrist and to integrate the efforts of the staff in carrying through the plans for individuals.

Finally, provision is made for the schooling of the children. School teachers live at Herrick House, and the children attend school in the building, or occasionally have their lessons at the bedside. This gives the child a chance to catch up with his class. At the same time there is no undue pressure put upon the child for school performance. Many of these children make up one or two semesters' work during their stay.

The present follow-up study is intended to evaluate the cardiac physical status of children one or more years after the beginning of their ambulation immediately after an acute rheumatic attack or recurrence. These children are designated in the study as immediate or recent convalescents.

The procedure employed in this study was as follows: Children who began ambulation at Herrick House more than one year previously were brought in for examination. At the time of examination they were questioned concerning their health during the interim period, their health in general, and specifically sore throats and other conditions which forced them to miss school; they were also asked whether or not they had received sulfadiazine or penicillin prophylaxis at any time. The physical examination consisted in a general observation and a check

for evidence of active rheumatic processes. Heart and lungs were thoroughly examined. A 2-meter chest roentgenogram was made. Fluoroscopy of the heart was carefully done by the method of Schwedel, whereby individual heart chambers were checked for size. An electrocardiogram was taken. This included the three standard limb leads, the Goldberger leads, and V chest leads, V_1 through V_7 and V_{4R}. Examination data were summarized and compared with the records of physical examinations carried out during the child's period of convalescence at Herrick House.

A control group, in a sense, was obtained by following up a number of rheumatic children who had spent a period at Herrick House as nonconvalescents. This includes children whose activity had been markedly restricted by their physicians or parents long after the attack of rheumatic fever had ended. Many of the children had been made into "cardiac cripples" by restriction and anxiety rather than by organic heart disease. Also included in the control group were cardiac children accepted at Herrick House for the summer vacation periods. These were well past the period of active rheumatic heart disease, and they represent a conglomerate group in which treatment during convalescence was not uniform. They serve as a "control" group only so far as they all had had rheumatic fever and engaged in ambulation at Herrick House long after the attack. Follow-up of these patients serves to give a partial picture of the natural history of the disease in the Middle West. Although all follow-up examinations were made by two physicians (A. J. S. and I. M.), the initial examinations were made by many other physicians. Such variation might introduce error into the study; however, so far as both control and immediate convalescent groups were examined in similar fashion, the results of the study would not be vitiated by such error. At the time of examination the physicians did not know which patients were in the "control" and which were in the immediate convalescent group.

A total of 239 patients were followed. Of these, 206 were available for comparative study; 12 had died since leaving Herrick House, and 21 could not be included in the series for a variety of reasons, discussed below.

Patients were classified as better, same, or worse at the time of follow-up depending upon the presence of significant heart murmurs and changes in the size of specific heart chambers as seen on fluoroscopy. The 2-meter chest roentgenogram was not as useful as fluoroscopy because in growing children the relationship of heart size to chest size is complex. Also, early chamber enlargement cannot be seen in the posteroanterior roentgenographic view. The electrocardiogram was not used for comparative purposes because large numbers did not have them taken during their first Herrick House admission, and also, different leads were used in the follow-up study.

A patient was classified as better when previously heard murmurs had disappeared and at the same time enlarged heart chambers had returned toward normal; if the reverse occurred, or new murmurs occurred, with or without additional enlargement, or if enlargement occurred either without changes or with apparent improvement in murmurs, he was classified as worse. Significant murmurs were considered to be any diastolic murmurs and systolic murmurs Grade 3 or more in intensity. Thus, the appearance of a diastolic murmur without changes in heart size classified the patient as worse.

Table 2 summarizes the clinical status of all patients investigated. They are classified as immediate convalescent or postconvalescent and are grouped according to time intervals since beginning ambulation, or, for postconvalescents, since beginning their Herrick House sojourn. There are 114 recent convalescents who are better or unchanged, and 36 who are worse. In the postconvalescent group there are 37 better or unchanged and 19 worse.

The number of patients investigated to date are too few for statistical treatment; however, the over-all picture is quite clear. Approximately 24% of recent convalescents are worse, as contrasted with 34% of nonconvalescents. Specifically, the data suggest that accelerated rehabilitation, as carried out at Herrick House, does not harm the heart.

This impression finds support in the work of Karpovich and associates.[2] These investigators found that a graded system of physical exercises was not harmful to the hearts of soldiers who had recovered from acute rheumatic fever. On the basis of observations made 6 to 12 months after graded exercise was begun, they reported no difference in the incidence of heart damage in three groups of patients for whom the average periods of quiescence had been 91.1, 77.3, and 16.2 days respectively.

TABLE 2.—*Clinical Status of 206 Patients Investigated*

Follow-Up Period, Yr.*	Immediate Convalescents (150)			Postconvalescents (Controls) (56)		
	Better, No.	Same, No.	Worse, No.	Better, No.	Same, No.	Worse, No.
1-1½	4	19	8	0	7	1
1½-2	4	20	8	1	4	0
2-3	6	33	9	0	7	6
3-4	1	23	11	0	9	8
Over 4	1	3	0	2	7	4
Total	16	98	36	3	34	19

* For immediate convalescents the follow-up period is measured from the time that they began ambulation; for postconvalescents, from the time that they began their Herrick House sojourn.

The worsening of the condition of a certain percentage of patients after rheumatic fever is evident from a perusal of studies in other areas. Stroud and others,[3] in Philadelphia, reporting on 307 patients cared for at a convalescent hospital, stated that 21% were dead at the end of 10 years. Only 182, or 59%, were working, able to work, or going to school. Coombs,[4] in Bristol, England, gives a similar figure, of 253 patients, 21.4% dead at the end of 10 years. Jones and Bland,[5] in a 10-year follow-up study on 1,000 cases of rheumatic fever in the Boston area, reported that of 342 patients classified as having potential heart disease, 27% were worse, and of 658 classified as having rheumatic heart disease, 51% had become worse, and of these, 192 were dead. Thus, of the 1,000 patients followed, at the end of 10 years, 43% had become worse. The studies of Wilson [6]

2. Karpovich, P. V.; Starr, M. P.; Kimbro, R. W.; Stoll, C. O., and Weiss, R. A.: Physical Reconditioning After Rheumatic Fever, J. A. M. A. **130**:1198 (April 27) 1946.

3. Stroud, W. D.; Goldsmith, M. A.; Polk, D. S., and Thorpe, F. Q.: Ten Years' Observation of Children with Rheumatic Heart Disease, J. A. M. A. **101**:502 (Aug. 12) 1933.

4. Coombs, C. F.: Rheumatic Heart Disease, New York, William Wood & Company, 1924.

5. Jones, T. D., and Bland, E. F.: Rheumatic Fever and Heart Disease: Completed 10-Year Observation on 1,000 Patients, Tr. A. Am. Physicians **57**:267, 1942.

6. Wilson, M. C.; Lingg, C., and Croxford, G.: Statistical Studies Based on the Classification of Heart Disease, Am. Heart J. **3**:196 (Dec.) 1928.

give a mortality rate of 11.5% at the end of 10 years. The roentgenological studies of Taussig [7] indicate that after an attack of rheumatic fever the heart may grow normally, or it may after a period of enlargement remain stationary in size while the chest grows, or it may enlarge progressively. It is in the group with the last type of change that the high mortality rate and invalidism occur. The reports of Taran [8] give a more favorable outlook for his patients, in that his mortality and recurrence rates are lower in the early years than those of other studies. All of these authors indicate that a worsening of the cardiac status of the rheumatic child is due to persistent, active infection or recurrences.

The criticism which may be leveled at all studies of the convalescent care in rheumatic fever is that we have no accurate method for indicating that the disease is inactive. After all laboratory evidence is considered, the decision as to when a child is ready for ambulation depends upon the judgment of the individual physician.

Stroud points out that his mortality figures are not so good as those of Wilson, yet her patients were those followed in clinics; they had not received the careful

TABLE 3.—*Changes in Patients Classified as "Worse"**

	Chamber Enlargement † Without Changes in Murmurs			Changes in Murmurs Without Changes in Chamber Size		Appearance of New Murmurs and Changes in Heart Size	Total
	LA	LV	2 or More Chambers	AI	MS		
Immediate convalescent group.....	11	3	8	4	1	9	36
Immediate convalescents with recurrences	0	1	4	2	0	7	14
Postconvalescent (control) group.....	5	4	4	2	0	4	19
Postconvalescents with recurrences.....	0	0	0	0	0	1	1

* LA indicates left atrium; LV, left ventricle; AI, aortic insufficiency; MS, mitral stenosis.
† Chamber enlargement signifies enlargement not previously present or progressive enlargement.

convalescent care which his were given at the hospital. In conclusion, he was not certain that the method of convalescence had much to do with the results.

Table 3 summarizes the findings on patients classified as worse in the present follow-up study. Of the nine immediate convalescents who had changes in both heart size and murmurs at the time of follow-up, seven had had intervening recurrences. Eleven immediate convalescents had only left atrial enlargement as a single defect, and in all patients the change was described as minimal or 1+. Five postconvalescent patients had left atrial enlargement as a single minimal defect. All 16 patients with minimal left atrial enlargement (both immediate convalescents and postconvalescents) were without recurrences.

While a 1+ backward bowing on the esophagram taken in the right anterooblique position with the patient in deep inspiration has frequently been considered definite evidence of left atrial enlargement, there has been no clear demonstration

7. Taussig, H. B., and Goldenberg, M.: Roentgenological Studies of the Size of the Heart in Childhood, Am. Heart J. **21**:440 (April) 1941.

8. Taran, L. M.: Treatment of Acute Rheumatic Fever and Rheumatic Heart Disease: Seminars on Rheumatic Fever and Rheumatic Heart Disease, Am. J. Med. **2**:98 (Jan.-June) 1942.

that this is necessarily true. Schwedel [9] states that in horizontal hearts there may be a normal slight backward bowing. Thus there may be a normal variation on the esophagram from absolutely straight to minimal backward bowing. While 2 + backward bowing on deep inspiration is probably definite evidence of left atrial enlargement, 1 + may be due to other causes, such as (1) normal variation, including that due to elevation of the diaphragm, obesity, and squat heart; and (2) old mediastinal disease. In this way, 1 + backward bowing may be compared with 1 + fullness of the middle salient (pulmonary artery segment). The real significance of cases with minimal changes on esophagrams will be shown in the years to come, when changes in the amount of bowing are more frequently recorded. It may be that a number of these patients should not have been placed in the "worse" group.

It is well known that rheumatic carditis may continue as a smoldering, subclinical disease, and this may be the factor responsible for the progression of the heart enlargement or changes in murmurs in most of our cases. In addition to the carditis there is the mechanical factor of stress and strain with the changing dynamics of progressive valvular defects. This is particularly well demonstrated

TABLE 4.—*Follow-Up Data According to Racial Groups and Sex*

	White Girls, No.	Negro Girls, No.	White Boys, No.	Negro Boys, No.	Total
Immediate Convalescents					
Better	6	2	5	3	16
Same	37	6	43	12	98
Worse	15	3	16	2	36
Postconvalescents (Controls)					
Better	0	0	3	0	3
Same	15	1	13	5	34
Worse	7	2	7	3	19
Total	80	14	87	25	206

in the right ventricular enlargement which follows mitral stenosis. The left atrium enlarges, followed by elevated pressure in the pulmonic circuit, pulmonary artery enlargement, and finally right ventricular enlargement. In contrast to this sequence are the dynamic changes of Lutembacher's syndrome. Here, with mitral stenosis the left atrium is frequently of normal size, whereas the right atrium is grossly enlarged. It is probable that the dynamic factors are more important in the older age groups. The present surgical approach to stenotic valvular disease is primarily an attack upon the mechanical factors involved.[10]

Table 4 gives a division of data according to sex and racial groups. Negroes constituted only a small segment of the whole follow-up group, 39 of 206 patients. This does not reflect difficulty in following the Negro children but rather parallels the proportion of Negroes who have convalesced at Herrick House.

Twenty-one patients could not be included in the follow-up study. Although many of these had been sent to Herrick House as suspected rheumatics, at the time of follow-up it was found that nine had congenital heart disease, two had

9. Schwedel, J. B.: Clinical Roentgenology of the Heart, Ann. Roentgenol. **18**:57,1948.

10. Glover, R. P.; Bailey, C., and O'Neill, T. J. E.: Surgery of Stenotic Valvular Disease of the Heart, J. A. M. A. **144**:1049 (Nov. 25) 1950.

pulmonary disease, one had a postviral myocarditis, one had sickle cell anemia, four were considered diagnostic problems, three were discovered to have active rheumatic fever and were returned to Chicago hospitals, and one was a rheumatic who remained at Herrick House only five days, so he was not considered part of our program.

There were 12 deaths in the follow-up group. Only two of these patients had received accelerated rehabilitation. These two had rheumatic recurrences, progressive failure, and death many months after leaving Herrick House. Of the other 10, none had been included in the accelerated rehabilitation program. Eight of these had active rheumatic carditis and were kept at bed rest on very restricted activity during their brief Herrick House stay. They were returned to Chicago hospitals for care; there death occurred from progressive failure. Of the other two patients, one had subacute bacterial endocarditis and one had an associated cyanotic congenital defect and had died unexpectedly while riding on a street car some months after leaving Herrick House.

SUMMARY AND CONCLUSIONS

From the present study it is not possible to determine at what point in the years following ambulation heart-size change occurred. This follow-up is in effect a "spot check" four years after Herrick House initiated its year-round program. A continuing follow-up project, begun with the work here reported, is now under way to document more carefully the effects of accelerated rehabilitation. The data presented here will serve as a pilot study for further work.

While these rough figures may seem to indicate that accelerated convalescence is beneficial to the heart, this conclusion is not warranted, because of the variable factors in the control group; yet they are suggestive that deterioration of the patient's cardiac status is not related to the method of convalescence.

Without question, accelerated rehabilitation is psychologically beneficial to the child. Emotional maturation is encouraged by the early return to a cultural environment approaching normal living.

It has become increasingly evident throughout this study that the significance of the 1 + bowing on esophagrams must be more carefully delineated.

An analysis of the electrocardiogram in rheumatic heart disease will be presented in a subsequent paper.

PREMATURITY IN THE NEGRO INFANT

Study of Patients Admitted to Geo. W. Hubbard Hospital Nurseries During Eleven-Year Period

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INTEREST is currently maintained at a high level in the concerted effort to reduce the frequency of premature births and to increase the survival rates of such infants once the problem is imminent. Much of the birth and fatality data presently available were derived from studies of hospital records, while the true incidence of prematurity in the general population remains unknown. Bain, Hubbard, and Pennell [1] point out that not only do we lack knowledge of how many babies are born prematurely, but also we do not have good information on how many of those born prematurely live. Reports based on unreliable criteria involving the gestation period defend a figure in the neighborhood of 5% of all births. In the more reliable studies, based on a birth weight of 2,500 gm. (5½ lb.) or less, the incidence is reported between 7 and 11%.[2] The recent practice of most states, however, of requiring birth weight data on birth certificates portends improvement in the collection of such statistical information for comparison on a national scale.

The frequency of prematurity is conceded to be greater among the poorer economic groups, and it is therefore generally higher among Negroes. Hess and Lundeen,[3] in referring to the many factors influencing the premature birth rate, include heavy lifting, overwork, physical exhaustion, and other strenuous activities commonly engaged in by the underprivileged. Brown, Lyon, and Anderson [4] indidate that illness in the mother is associated with a decided increase in the rate of premature birth and that this effect is more prominent when observed in Negroes.

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Public Health Nurse for the Premature Station (Clara Wilson-Webb) and Statistical Clerk for the Pediatric-Pedodontia Program (Martha B. Pointer).

1. Bain, K.; Hubbard, J. P., and Pennell, M. Y.: Hospital Fatality Rates for Premature Infants, Pediatrics **4**:454, 1949.

2. Dunham, E. C.: Premature Infants: A Manual for Physicians, Federal Security Agency, Children's Bureau, Publication No. 325, 1948, pp. 13, 15, and 42.

3. Hess, J. H., and Lundeen, E. C.: The Premature Infant, Philadelphia, J. B. Lippincott Company, 1941, p. 3.

4. Brown, E. W.; Lyon, R. A., and Anderson, N. A.: Causes of Prematurity: IV. Influence of Maternal Illness on the Incidence of Prematurity, Am. J. Dis. Child. **70**:317 (Nov.-Dec.) 1945.

Dunham [2] suggests that the difference in the proportions of white and Negro infants weighing less than 2,500 gm. may not be racial. She points out that difference in economic status with accompanying differences in nutrition and complications of pregnancy may be responsible for the lower average birth weight reported for Negro infants.

Most studies on the broad problem of prematurity have included data on nonwhite subjects. Few, however, have been devoted largely or entirely to material derived from a Negro population specifically.

MATERIAL

The present study represents a review of the clinical records of all infants born in the Geo. W. Hubbard Hospital during the 11-year interval between January, 1940, through December, 1950, to determine the incidence of prematurity in an exclusively Negro population. In addition, because a new premature station was established in the pediatric service in 1948, an evaluation of the specific influence of these improved facilities on the survival data of such infants seemed pertinent. All infants under consideration were liveborn, and except for 67 patients admitted

TABLE 1.—*Incidence of Prematurity*

	Total Live Born, No.	Premature	
		No.	%
1940-1947	2,633	255	9.7
1948-1950	2,364	309	13.1
1940-1950	4,997	564	11.3

from surrounding communities, all were delivered in the Geo. W. Hubbard Hospital. Since 1948, through the support of the Tennessee State Health Department and the United States Children's Bureau, a well-equipped jeep-ambulance has been available for transportation of out-born premature infants to and from the hospital. Through these agencies, also, a public health nurse has been employed to insure adequate field nursing service and follow-up.

The definition of prematurity observed herein was based on the resolution adopted by the American Academy of Pediatrics in 1935: [5] "A premature infant is one who weighs 2,500 grams or less at birth (not at admission) regardless of the period of gestation."

The heterogenous socioeconomic background of subjects involved in this study varied from the lower echelons of the common laborers to the upper professional brackets, including families of college teachers, physicians, and lawyers.

INCIDENCE

Among nearly 5,000 live births in the Geo. W. Hubbard Hospital during the period of 1940-1950, there were 564 (11.3%) premature infants. Since the establishment of the premature station in 1948, of all infants born in the hospital, 13.1% weighed 2,500 gm. or less (Table 1).

5. Round-Table Discussion on Prematurity, in Proceedings, Fifth Annual Meeting of the American Academy of Pediatrics, New York, June 7, 1935, J. Pediat. **8**:117, 1936.

Peckham,[6] in a large series of infants delivered in the Johns Hopkins Hospital between the years 1896 and 1936, found that of those weighing less than 2,500 gm. at birth, 11.95% were "colored," while only 6.1% were white. More recently, Eastman [7] presented a statistical analysis of data of premature births at the same hospital for the period between 1926 and 1947. He found that of all infants born, premature infants constituted 11.7%.

In a recent report on data acquired from about half of the hospitals in the United States caring for 50 or more premature infants a year, Bain, Hubbard, and Pennell [1] found that of the liveborn infants weighing 2,500 gm. or less, 5.6% were white and 9.5% nonwhite.

Brockway, Reilly, and Rice,[8] in a study of 3,872 patients born at Kings County Hospital, Brooklyn, found the strikingly high incidence of prematurity of 15.5% for whites and 20% for Negroes.

Because the Geo. W. Hubbard Hospital premature station accepts newborn infants from adjacent communities, the total number of premature infants that received care in the nursery during the 11-year period was 631.

BIRTH WEIGHTS

Birth weights of these infants may be listed as follows:

	No.	%
Below 750 gm.	17	2.7
750-1,000 gm.	22	3.5
1,001-1,250 gm.	28	4.4
1,251-1,500 gm.	42	6.6
1,501-2,000 gm.	153	24.3
2,001-2,500 gm.	369	58.5

The great majority (82.8%) of these infants weighed more than 1,500 gm. at birth. Only a small number (6.2%) are included in the group weighing 1,000 gm. or less. This distribution of premature infants according to weight is similar to that for the 7,291 cases reported by Duffield, Parker, and Baumgartner [9] for New York City.

SEX

Boys and girls were almost equally divided (1:1.16). (Sex was not listed in four instances.)

	No.	%
Boys	291	46.4
Girls	336	53.6

FATALITY RATES

In order to evaluate the effect of improved nursing care and nursery facilities in reducing the hazards of prematurity in the Geo. W. Hubbard Hospital, a com-

6. Peckham, C. H.: Statistical Studies on Prematurity: I. The Incidence of Prematurity and the Effect of Certain Obstetric Factors, J. Pediat. **13**:474, 1938.

7. Eastman, N. J.: Prematurity from the Viewpoint of the Obstetrician, Am. Pract. **1**:343, 1947.

8. Brockway, G. E.; Reilly, E. T., and Rice, M. M.: Premature Mortality, J. Pediat. **37**:362, 1950.

9. Duffield, T. J.; Parker, S., and Baumgartner, L.: Birth Weight and Its Relation to Neonatal Mortality: An Analysis of Data on Live Births in New York City in 1939, Child **5**:125, 1940.

parison of fatality rates for the period before and after the establishment of the new premature station is presented in tabular form (Table 2).

Of 631 infants studied, 140, or 22.2%, died. When the two periods 1940-1947 and 1948-1950 are compared, it is observed that since the establishment of the pre-

TABLE 2.—*A Comparison of Fatality Rates Before and After Establishment of New Premature Station*

Weight Group	1940-1947			1948-1950		
	No. Births	Deaths		No. Births	Deaths	
		No.	%		No.	%
Below 750 gm.	3	2	66.7	14	14	100.0
750-1,000 gm.	10	10	100.0	12	8	66.7
1,001-1,250 gm.	13	10	76.9	15	4	26.7
1,251-1,500 gm.	14	10	71.4	28	12	42.9
1,501-2,000 gm.	65	25	38.4	88	20	22.7
2,001-2,500 gm.	153	13	8.5	216	12	5.6
Total	258	70	27.1	373	70	18.8

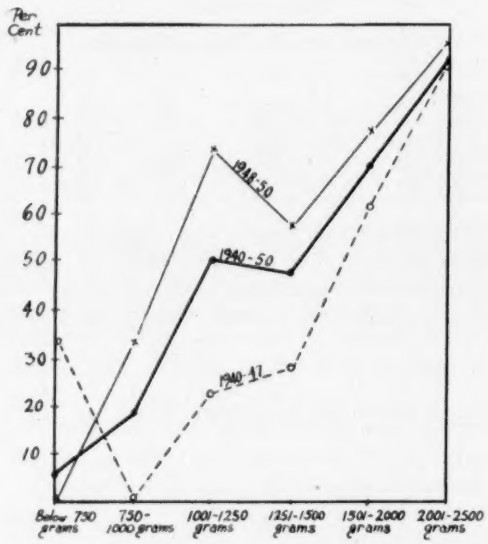

Chart 1.—A comparison of survival rates for the two periods of 1940-1947 and 1948-1950.

mature station in 1948 the fatality rate has been reduced by 8.3%. Chart 1 depicts the improvement in survival rates for these two periods. Results are particularly gratifying in the 750 to 1,500 gm. range.

Koch, Weymuller, and James [10] found that by improving the methods of caring for premature infants at the Long Island College Hospital, it was possible to reduce a previous fatality rate of 28.4% to a new level of 16.3%.

10. Koch, L. A.; Weymuller, C. A., and James, E.: Reduction of Mortality from Premature Birth, J. A. M. A. **136**:220 (Jan. 24) 1948.

RELATION OF RACIAL GROUPING TO FATALITY INCIDENCE

The strong plea for clarity, uniformity, and consistency in reporting data relating to the premature problem is vigorously supported. However, the present custom of employing the term "nonwhite" would seem to provide an additional opportunity for confusion to those interested in more specific information for purposes of comparison. It is a reasonable surmise that the category "nonwhite" includes Negro subjects predominantly, particularly when data from southern sections of the country are under consideration. This notwithstanding, however, the question naturally arises: "What percentage of 'nonwhite' is Negro? Mexican? Indian? or other?"

Plattner, Stein, and Gerber [11] studied a series of 1,660 nonwhite and 1,247 white premature infants seen in the Cook County Hospital. The fatality rates were listed as 15.7% and 20.6% respectively. The excess in death rate for whites was said to be dependent on the fact that only 16% of the infants born at Cook County Hospital

TABLE 3.—*Fatality Rates Among Premature Infants by Birth Weight Groups:* * *A Comparison of Data from Eight Hospitals* †

Name of Hospital	Dates	No. of Premature Infants	Fatality Rate, %				
			Less than 1,000 Gm.	1,000-1,500 Gm.	1,501-2,000 Gm.	2,001-2,500 Gm.	Total 2,500 Gm. or Less
Sarah Morris Hospital for Children, Chicago	1940-1945	1,993	91.3	50.0	14.4	7.2	25.8
Boston Lying-In Hospital	1943-1945	481	100.0	46.7	12.8	4.4	15.0
Johns Hopkins Hospital, Baltimore, Obstetric Department	1941-1945	904	....‡	48.8	16.8	4.2	....‡
Charity Hospital of Louisiana, New Orleans	1944-1945	1,122	95.7	55.4	30.5	7.9	27.4
New York Hospital, Pediatric Department	1943-1945	506	85.0	49.3	21.4	5.6	22.3
Long Island College Hospital, Brooklyn, Pediatric Department	1940-1945	635	90.0	41.4	18.3	4.6	16.1
Vanderbilt University Hospital, Nashville, Tenn., Pediatric Department§	1944-1950	650	96.1	66.1	17.9	4.8	20.2
Geo. W. Hubbard Hospital, Nashville, Tenn. (New Premature Station)	1940-1950 (1948-1950)	631 (373)	87.2 (84.6)	51.4 (37.2)	29.4 (25.0)	6.8 (5.6)	22.2 (18.8)

* Some of the hospitals used the weight groups 1,000-1,499, 1,500-1,999, and 2,000-2,499 gm.

† Data are adapted from Dunham.[2]

‡ Infants weighing less than 1,000 gm. are not included. Total rate is therefore not comparable with those of the other hospitals.

§ Data for Vanderbilt University Hospital were given by Dr. Amos U. Christie in a personal communication to the authors.

were white, whereas they constituted 76% of the premature infants transferred to the hospital from outside.

Bain, Hubbard, and Pennell [1] studied fatality data from 49 hospitals in the United States and found a rate of 24.5% for whites and 27.7% for nonwhites. Table 3 presents data from eight hospitals for scrutiny. Though not strictly comparable, these data serve the useful purpose of providing an opportunity to evaluate accomplishment at the Geo. W. Hubbard Hospital in terms of results achieved in the larger centers.

INFLUENCE OF SEX ON FATALITY RATES

Although female births slightly outnumber male births, a significant preponderance of male deaths is to be noted (Table 4).

11. Plattner, E. B.; Stein, A. F., and Gerber, H. X.: Analysis of Premature Mortality Rates, Pediatrics **5**:194, 1950.

Abrahamson [12] pointed out that "the constantly higher [fatality] rates among the male babies are quite apparent, and again demonstrate the universal phenomenon of excess mortality among males as compared with females. While the frequency of premature births is greater in female babies, probably because of their smaller birth weight and birth length, a much higher stillborn ratio and death rate occur in male infants." Abrahamson refers to the theory of Lenz,[13] which has been supported by others,[14] in which the excess mortality observed in males is explained on the basis of the operation of recessive hereditary sex-linked factors.

TABLE 4.—*Fatality Rates by Sex and Birth Weight, 1940-1950*

Weight Groups	No. Births	Male Deaths		Female Deaths	
		No.	%	No.	%
Below 750 gm.	17	9	55.6	7	44.4
750-1,000 gm.	22	13	82.2	5	17.8
1,001-1,250 gm.	28	8	57.1	6	42.9
1,251-1,500 gm.	42	10	45.5	12	54.5
1,501-2,000 gm.	153	28	62.2	17	37.8
2,001-2,500 gm.	369	20	80.0	5	20.0
Total	631	88	62.9	52	37.1

TIME OF DEATH

Of the 140 fatalities, more than one-half occurred during the first 24 hours:

	%
Within first hour	11.4
Within first 24 hours	55.1
Within first month	93.0

These data would seem to indicate that the premature death rate is of relative insignificance after the first month of life. This is consistent with the clinical observation that most of the inherent hazards of prematurity have already been encountered during this early critical 30-day period.

Abrahamson,[12] in relating the factor of postnatal age to fatality rates, indicated that death occurs during the first 24 hours after delivery in about 55 to 60% of cases of premature birth. A national survey [15] for 1948 demonstrated that of the premature infants who died, 57% died during the first day, and 98% had died by the end of the first month.

Chart 2 reveals that in the present study, of those infants who died in the group weighing less than 1,000 gm., 82.4% died within the first 24 hours; while 50% of those weighing 1,001 to 1,500 gm., 46.6% of those between 1,501 and 2,000 gm., and 40% of those between 2,001 and 2,500 gm. died during the same interval.

12. Abrahamson, H.: Factors Influencing Premature Infant Mortality, J. Pediat. **19**:545, 1941.

13. Lenz, F.: Die Uebersterblichkeit der Knaben im Lichte der Erblichkeitslehre, Arch. Hyg. **93**:126, 1923.

14. Schirmer, W.: Ueber den Einfluss geschlechtsgebundener Erbanlagen auf die Säuglingssterblichkeit, Arch. Rassen-u. Gesellsch.-Biol. **21**:353, 1929.

15. Mortality from Premature Birth and Associated Causes of Death, Federal Security Agency, Children's Bureau, Statistical Series No. 5, 1948, p. 1.

It is of interest to note, in the same chart, that with the increase in birth weight, there also occur a consistent increase in deaths after the first month of life and a concomitant decline in fatalities during the first hour of life. An analysis of the causes of death in this later period would doubtless reveal similarities with causes of death in normal newborn infants in the same postnatal age group.

A total of 67 premature infants were admitted to the hospital during the period of this study:

	No.	%
1940-1947	3	4.5
1948-1950	64	95.5

Only 59 records, however, were sufficiently complete to be included in this phase of the report (Table 5).

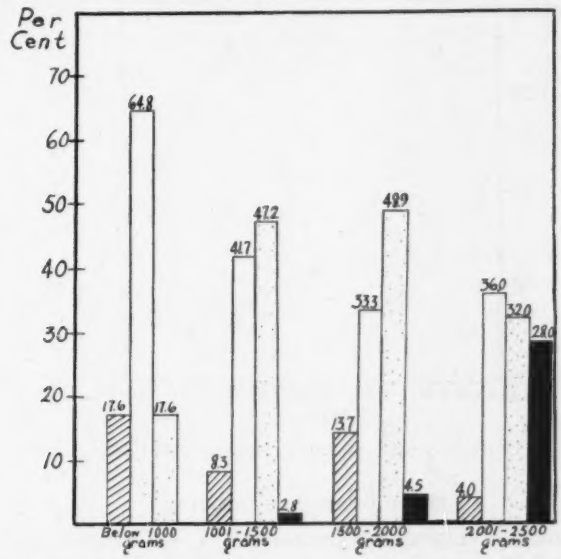

Chart 2.—Time of death by weight groups for 1940-1950; the lined bar indicates deaths occurring within the first hour after birth; the solid white bar, deaths within the first 24 hours after birth; the dotted bar, deaths within the first month after birth, and the solid black bar, deaths later than one month after birth.

TABLE 5.—*Fatality Rates of Infants Admitted to Hospital After Birth Outside, 1940-1950*

Weight Groups	No.	Deaths	%
Below 750 gm.	5	5	100.0
750-1,000 gm.	2	2	100.0
1,001-1,250 gm.	7	1	14.3
1,251-1,500 gm.	6	2	33.3
1,501-2,000 gm.	22	5	22.7
2,001-2,500 gm.	17	2	11.8
Total	59	17	28.8

It is to be noted here that the total fatality rate is higher than that observed in the case of patients born in the hospital (22.2%) during the same 11-year period.

If the comparison is adjusted, however, to account for excessive heaping of outside admissions (95.5%) toward the 1948-1950 period, the contrast is more striking:

	%
Out-born fatality rate (1948-1950)	26.8
In-born fatality rate (1948-1950)	18.8

Thus, the excess in infant deaths for outside admissions, under these circumstances, is 8%. Experience has shown that this excessive death rate is dependent upon exposure of infants in transit to the hospital, as well as upon inadequate care during and immediately after delivery. Delay in seeking hospital care is an additional prominent contributing factor in boosting the fatality rate. In this extramural group it is to be noted that no infant weighing below 1,000 gm. survived.

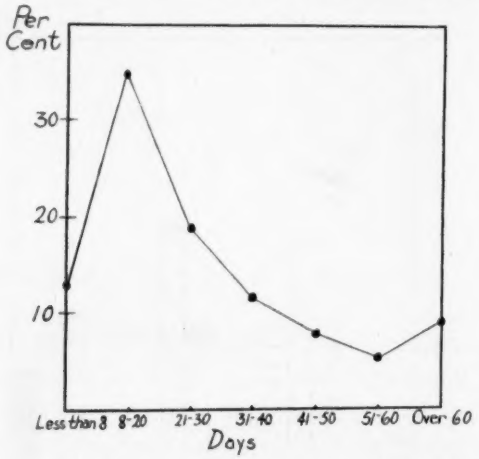

Chart 3.—Length of stay in hospital, 1940-1950.

LENGTH OF STAY IN HOSPITAL

Data on duration of hospital stay are available on 443 of the infants who survived. Of these 65.2% were discharged during the first month of life, while 15.2% remained more than 50 days (Chart 3).

Wallace, Baumgartner, and Park [16] reviewed the American literature and found that the average length of hospital stay of premature infants in a series which consisted only of those premature infants who survived ranged from 24 to 28 days. They reported an average duration of 34.2 days of hospitalization for another group of surviving premature infants for whom the Federal Emergency Maternity and Infant Care Program underwrote costs of hospital care.

Inasmuch as the Geo. W. Hubbard Hospital premature station has provided premature care since 1948 practically without cost to the family, the economic element of ability to pay has not been a deciding factor on length of hospital stay. This notwithstanding, data at hand reveal no significant change in duration of hospitalization attributable to newer facilities.

16. Wallace, H. M.; Baumgartner, L., and Park, M. L.: The Average Length of Stay in the Hospital of Infants Born Prematurely, Pediatrics **1**:66, 1948.

AGE OF MOTHER

The ages of 539 mothers were available for study. More than one-fourth (28%) of all mothers were less than 20 years of age, while 83% were below 30 years. Only 2.6% were found in the group above 40 years of age (Chart 4).

An attempt was made to determine if the age of the mother provided any specific influence upon the incidence of premature birth. The ages of all mothers who gave birth to normal newborn infants in the Geo. W. Hubbard Hospital [17] during the period of this study were ascertained. As is observed in Chart 4, no difference could be demonstrated between the general maternal population and the premature maternal group. That is to say, in any given group of 100 infants, whether full term or premature, born at the Geo. W. Hubbard Hospital, one would expect to

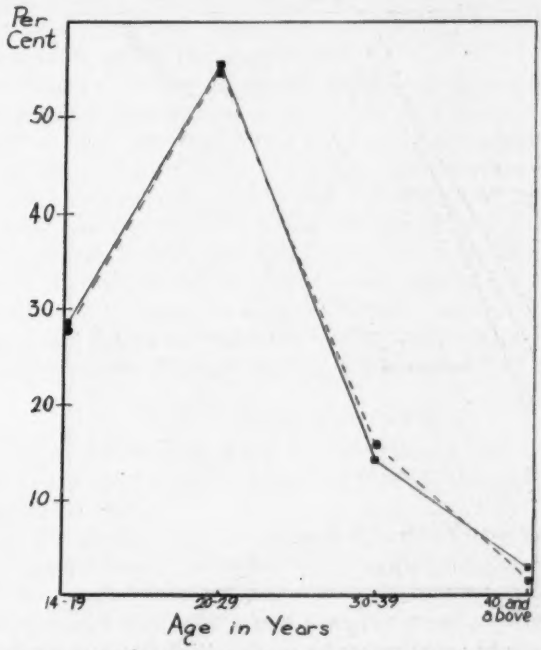

Chart 4.—Birth rate by age of mother for the Geo. W. Hubbard Hospital for the period 1940-1950: rate for normal newborn infants (4,527 cases) is indicated by the broken line; for premature infants (631 cases), by the solid line.

find about 27 or 28 with mothers between 14 and 19 years of age, 54 or 55 with mothers between 20 and 29 years of age, about 14 to 16 with mothers between 30 and 39 years of age, and between 1 and 3 with mothers 40 years of age and above.

With data on live births for Tennessee, supplied by Dr. Ruth R. Puffer,[18] statistician of the Tennessee Health Department, Chart 5 was constructed to ascertain the possible existence of any correlation between the maternal age groups for premature infants of this study and those for liveborn Negro and white infants of the state of Tennessee.

17. All maternal ages were not available.

18. Puffer, R. R.: Personal communication to the authors.

It is immediately apparent that the frequency of Negro live births in the general population of Tennessee exceeds that of whites only in the age group below 20 years. The correlation between premature birth rates (by age of mother) and Negro live births for the state of Tennessee, though not so close as is seen in Chart 4 for hospitalized subjects exclusively, is much closer than that for whites. In general, however, it is permissible to state that the incidence of premature birth is higher for hospitalized Negro mothers below 30 years of age than it is for Negro live births in the general population in Tennessee, and that this condition is reversed when the maternal age exceeds 30 years.

Tyson [19] found that 59% of almost 3,000 mothers of the premature infants he studied were between the ages of 20 and 30 years. This maternal age group con-

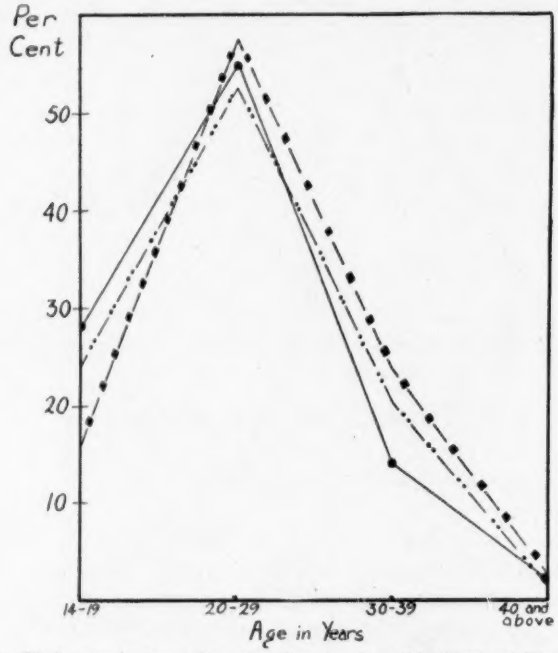

Chart 5.—Birth rate by age of mother for the Geo. W. Hubbard Hospital and the state of Tennessee: rate for premature infants (631 cases) from the Geo. W. Hubbard Hospital is indicated by the solid line with dots; for Negro newborn infants (15,091 cases) born in Tennessee in 1949, by the broken line with dots; for white newborn infants (66,773 cases) born in Tennessee in 1949, by the broken line with squares. The data for the state of Tennessee included only ages 15 years and above.

stitutes 55.1% in the present study; thus the findings of Tyson are closely approximated. Peckham [20] reported that 33.7% of the deliveries in the obstetrics service at Johns Hopkins were for women below 20 years of age. In addition, this group gave birth to 37.7% of the infants weighing less than 2,500 gm. and 30% of those measuring less than 45 cm. in length. In consideration of these findings, he sug-

19. Tyson, R. M.: A 15-Year Study of Prematurity from the Standpoint of Incidence, Mortality, and Survival, J. Pediat. **28**:648, 1946.

20. Peckham, C. H.: I. Statistical Studies on Prematurity, J. Pediat. **13**:474, 1938.

gests that early childbearing does not increase the incidence of true premature birth, but rather that the young woman at term gives birth to an infant smaller in weight but equal in length to the more mature newborn infant.

PRENATAL CARE

Of 591 mothers on whom data are available, 524 (88.7%) had either clinical or private prenatal care. No specific statement can be made, however, regarding the duration or quality of this care, except to point out that 70.2% were seen one or more times in the Geo. W. Hubbard Hospital prenatal clinic during the period 1940-1950. Prenatal care of some description was reported by 82.3% in the interval of 1940-1947, while 92% had such supervision during 1948-1950. Despite this apparent improvement in maternal prenatal care, the incidence of prematurity has increased by 3.4% since 1948. Apart from excursions in speculation, no data are presently at hand to explain this observation.

Eastman [7] found a striking correlation between the high incidence of prematurity and inadequate prenatal care. In 2,270 cases of single pregnancy with spontaneous onset of labor, the frequency of prematurity for women with good care was 7.8%, as compared with 24.9% for women with poor care or no care at all. Stander [21] cites Laurié, who has shown that the duration of pregnancy depends, in part, upon the extent to which the patient can spare herself during the last three months of pregnancy. Laurié found that pregnancy was prolonged an average of 20 days longer in 1,550 women who lived comfortably in a hospital for several months prior to delivery than in an equal number of women who entered at the onset of labor. Her figures emphasize the fact that hard work in poorly nourished women predisposes to the premature ending of pregnancy.

SUMMARY AND CONCLUSIONS

A review of records of nearly 5,000 live births at the Geo. W. Hubbard Hospital for the 11-year period 1940 through 1950 reveals that prematurity occurred in 564 instances (11.3%).

Since the establishment of a new premature station in 1948, the incidence of prematurity has risen from 9.7% to 13.1%, an increase of 3.4%. This unexplained rise in rate was not associated with significant fluctuations in the local economy, and it occurred despite a reported rise in the incidence of prenatal care.

Of 631 infants studied for the 11-year period (67 admitted from outside), 140, or 22.2%, died. Since the establishment of the premature station in 1948, however, the fatality rate has been reduced by 8.3%.

Over half (55.1%) of the deaths occurred within the first 24 hours of life. More than twice as many (82.4%) of the small infants (less than 1,000 gm.) died on the first day of life as did the larger infants (40%) weighing over 2,000 gm.

Sex exerts no significant influence on the incidence of premature birth. The ratio of male deaths to female deaths, however, was almost 2:1.

The fatality rate for out-born premature infants was 28.8%, as compared with 22.2% for in-born subjects for the same period. Although 95.5% of the former were admitted since the establishment of the new premature station, it has not been

21. Stander, H. J.: Williams Obstetrics: A Textbook for the Use of Students and Practitioners, Ed. 8, New York, Appleton-Century Company, Inc., 1941, p. 259.

possible to reduce the fatality rate more than 2% in this group. Some of the foremost factors responsible for this fixed high fatality rate in out-born infants are delay in seeking hospital admission and improper care prior to hospitalization.

The respective maternal age groups for premature infants in this study were practically identical in size with those for mothers of normal infants born in the Geo. W. Hubbard Hospital. From these data, it should be possible to predict with some degree of accuracy that for any group of 100 premature or otherwise normal infants born in this hospital, one would expect to find about 27 or 28 with mothers between 14 and 19 years of age; 54 or 55 with mothers between 20 and 29 years of age; 14 or 16 with mothers between 30 and 39 years of age, and 1 or 2 with mothers 40 years of age and above. Data for white liveborn infants for the state of Tennessee, when compared with data from the Geo. W. Hubbard Hospital, indicate significant racial differencies in regard to the size of the respective maternal age groups.

Although the number of women reporting prenatal care of some description increased by 10% since 1948, no reduction in the incidence of prematurity has ensued.

Mrs. Myrtle Senior-Adams and Mrs. J. M. Moore helped with the assemblage of the original data.

Case Reports

ACUTE RETICULOENDOTHELIOSIS (LETTERER-SIWE DISEASE)

Report of a Case in Which Several Unusual Features Are Combined

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AND
BERT E. STOFER, M.D.
WICHITA, KAN.

SINCE Letterer's description 24 years ago,[1] about 30 to 35 cases of acute nonlipid reticuloendotheliosis have reached the literature. Reticuloendothelial disease of this type has not been completely defined because of the small number of cases.[2]

We have recently encountered a case of fatal reticuloendotheliosis in which were combined a number of the less frequently mentioned features of this disease. It supports the idea of Wallgren[3] and others that the whole group represents varying degrees of pathologic change in the reticuloendothelial system.

REPORT OF A CASE

A white, 16-month-old boy entered Wesley Hospital on March 16, 1950, with a chief complaint of pallor, fever, and loss of weight. This child was a full-term infant with normal delivery, weighing 7 lb. 6 oz. (3,350 gm.) at birth. He was breast-fed until 9 months of age and in good health until that time. The mother had no illness during pregnancy. There were no other children in the family. The family history revealed arrested tuberculosis in the maternal grandmother. There was no history of rheumatic fever, diabetes, heart trouble, kidney disease, epilepsy, hay fever, urticaria, cancer, liver diseases, intestinal diseases, or mental diseases. This child had been receiving a multiple vitamin supplement since birth. He had always eaten well and was eating well on admission. His diet consisted of milk, cereal, fruits, vegetables, and eggs. At 9 months of age he had an upper respiratory tract infection accompanied with otitis media. The upper respiratory tract infection was treated with penicillin and local medication. The ear infection, however, did not clear up. Shortly after the upper respiratory tract infection the child began to have a scalp rash. A dermatologist diagnosed this as seborrheic dermatitis. It had been quite resistant to treatment and was present on entrance. Four months before entry, at 12 months of age, the child weighed 21 lb. (9,530 gm.), but he had gained no weight since that time. Two months previous to entry the child began to have some distention of the abdomen after ingestion of food. During the last month a definite and persistent enlargement of the abdomen had been noticed. Three weeks prior to entry the child had a slight yellowish mucous diarrhea, with as many as six to seven stools a day. This was checked by a diet of apple powder and skimmed milk. He did not vomit at any time. During the last two weeks

From the Wichita Foundation of Medical Research.

1. Letterer, E.: Aleukämische Retikulose, Ztsch. Path. **30**:377-394, 1924.

2. (*a*) Havard, E.; Rather, L. J., and Faber, H. K.: Nonlipoid Reticuloendotheliosis (Letterer-Siwe's Disease), Pediatrics **5**:474-485 (March) 1950. (*b*) Schafer, E. L.: Nonlipid Reticulo-Endotheliosis: Letterer-Siwe's Disease, Am. J. Path. **25**:49-73 (Jan.) 1949. (*c*) MacKelvie, A. A., and Park, W. W.: Letterer-Siwe Disease, Arch. Dis. Childhood **24**:93-98 (March) 1950. (*d*) Orchard, N. P.: Letterer-Siwe's Syndrome: Report of a Case with Unusual Peripheral Blood Changes, ibid. **25**:151-155 (June) 1950. (*e*) Claireaux, A. E., and Lewis, I. C.: Reticuloendothelial Granuloma: A Review with a Report of a Case of Letterer-Siwe Disease, ibid. **25**:142-150 (June) 1950.

3. Wallgren, A.: Systemic Reticuloendothelial Granuloma, Am. J. Dis. Child. **60**:471-500 (Sept.) 1940.

the urine had been rather dark. The child had had no contagious diseases; he had received immunizations for pertussis, diphtheria, and tetanus. There was no history of any convulsions or paralysis.

Physical examination revealed an undernourished, marasmic, slightly underdeveloped, very pale, irritable male infant. The skin had a waxy appearance, and there were old petechial hemorrhages on the chest, abdomen, and groin. There was a seborrheic dermatitis of the scalp. A slight icteric tinge to the sclera was present. In both external canals there was a purulent discharge. The ear membranes were perforated bilaterally. Shotty cervical and inguinal nodes were found. Rigidity and tenderness with pain on flexion of the neck were demonstrated. The liver was three fingerbreadths below the costal margin and was smooth, with rounded lower margin. There was also a mass in the left upper quadrant, separate from the liver and extending 4 cm. below the costal margin. It was mobile and tender to touch. Numerous ischial sinuses with purulent exudate were seen on rectal examination. There was generalized weakness and emaciation of the extremities. The Kernig and Brudzinski signs were present. No other significant findings were present.

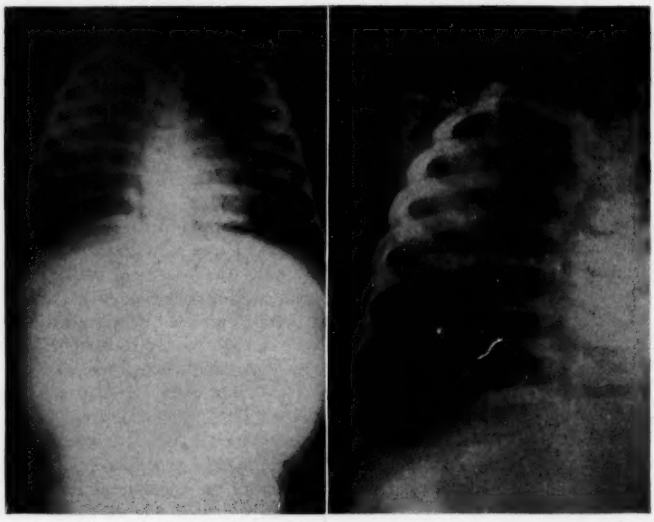

Fig. 1.—Left, diffuse miliary infiltration of the lungs shown in roentgenogram taken on sixth hospital day; right, enlargement of upper left portion of the roentgenogram, showing emphysema of mediastinum, neck, and chest wall.

A flat roentgenogram of the chest taken March 16 showed a diffuse miliary infiltration throughout both lungs. The heart, diaphragm, and mediastinal shadows appeared normal.

The impression of the roentgenologist, Dr. N. C. Nash, was that the process represented miliary tuberculosis.

Reexamination on March 22 showed no essential change in the extent or type of the infiltration (Fig. 1, left). There was, however, considerable subcutaneous emphysema in the neck and the lateral chest walls (Fig. 1, right). Roentgenological survey of the osseous system showed no recognizable alterations.

The principle deviations from normal in the laboratory examinations on entry were as follows: red blood cells, 3,650,000; white blood cells, 10,200; hemoglobin, 11.0 gm.; polymorphonuclear leucocytes, 27%; lymphocytes, 62%; mononuclear cells, 11%; thrombocytes (platelets), 70,000; trace of albumin in the urine; sedimentation rate, 23 mm. in one hour (Wintrobe); blood cholesterol, 496 mg. per 100 cc.; icterus index, 5 units; blood in stool (3+); aural exudate positive for staphylococci.

During the course of hospitalization the anemia and leucocytosis persisted at the same level. The relative lymphocytosis reverted toward normal for the age. Terminally 2% plasma cells

and 4 nucleated red cells per 100 white cells appeared in the peripheral blood. The count on March 23, one week after admission, was polymorphonuclear leucocytes, 50%; lymphocytes, 46%; monocytes, 2%. Cell count in the spinal fluid on admission was 50 lymphocytes and 12 neutrophiles. On two other occasions counts of 80 lymphocytes and 20 neutrophiles and 29 lymphocytes and 7 neutrophiles were reported. The icterus index increased to 60 units terminally.

The important negative findings were for the Klein test, blood and spinal fluid culture for pyogenic and mycotic infections, bone marrow, aural and perineal drainage for fungi, and intracutaneous tests with tuberculin and histoplasmin. The bone marrow showed a normal differential count and no abnormal forms. Guinea pig and special media inoculations for tuberculosis were negative.

The child continued to eat well for the first three or four days of hospitalization, but there was a progressive increase in the size of the spleen and the liver. On the fifth day of hospitalization a slight jaundice was noticed. On the sixth day a biopsy of the left inguinal node was

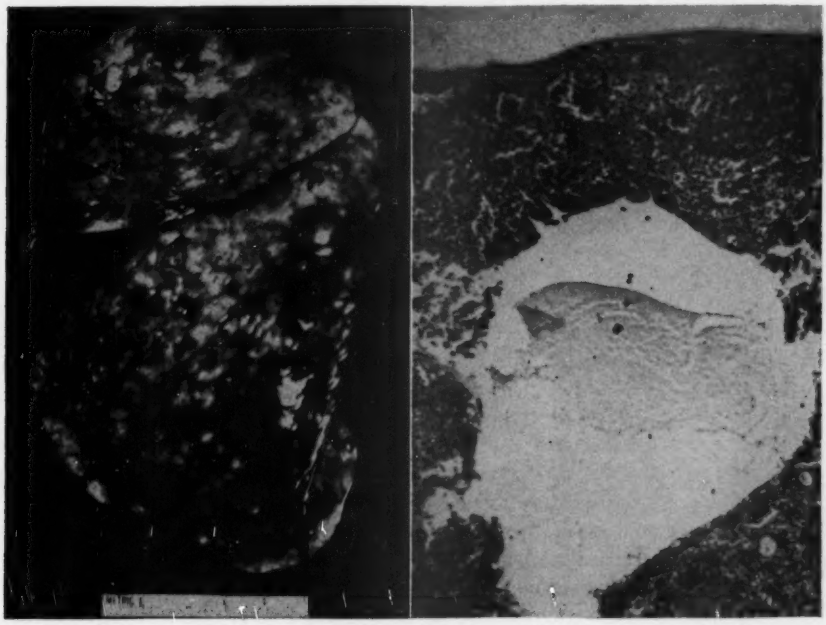

Fig. 2.—Left, external surface of the lung, showing multiple subpleural cysts and small emphysematous blebs; right, one of the subpleural cysts, with secondary atelectasis in the surrounding area (low power).

done. In the afternoon subcutaneous emphysema was noticed over the tissues of the neck. The child had no increase in nuchal rigidity but became more irritable and lethargic. Breathing slowly became more difficult, but no change in the character of breath sounds was noticed at any time. The ears and rectal fistulae continued to drain throughout the last days of life. There was no improvement in the seborrhea of the scalp. The rash showed no regression. The petechiae became darker and more numerous; new ecchymoses were seen each day. By the last day of life the child had become considerably dyspneic and irritable. Terminally there were a large protuberant abdomen, subcutaneous emphysema over the entire chest, and emaciation of the extremities. The waxy yellow color of the skin continued, and there developed new petechiae, as well as scattered ecchymoses. The infant was given dihydrostreptomycin, 0.25 gm., intramuscularly, twice daily. Two transfusions of fresh whole blood, 100 cc., were administered to reduce the bleeding tendency. Acute respiratory distress developed, and the patient became markedly cyanotic terminally.

At autopsy the important findings externally were a crusting seborrhea of the scalp, draining ears, emphysema subcutaneously present in the neck, petechial hemorrhages widely distributed over the body, and distention of the abdomen. In the thorax the lungs were strikingly involved by foci of grey-white consolidation. Many of the foci were associated with small cysts or tiny emphysematous blebs (Fig. 2). There was enlargement of lymph nodes, both peripheral and in the body cavities. The liver and spleen both were enlarged, the latter most markedly. The middle ears were involved in a purulent process. Recent infarcts were present in the liver.

Microscopic examination showed two principal cell types. The first was a large round oval cell with an oval vesicular, or occasionally reniform, nucleus, sharp nuclear border, and indistinct amphoteric cytoplasm. These were occasionally found in multinucleated form. The second cell was larger, irregular in outline, with a homogeneous, fairly dense, nuclear pattern and a vacuolated cytoplasm. In some tissues a phagocytic cell containing brown pigment was associated with the other cell types. In other areas eosinophiles were present, occasionally in fairly large numbers. These differences are tabulated below:

Organ Involved	Reticulo-endothelial Cells	Vacuolated Cells	Eosino-philes	Pigment Phago-cytes
Skin	+	0	0	0
Spleen	+	+	0	+
Bone marrow	+	+	+	0
Lung	+	+	0	0
Thymus	+	+	0	+
Nodes	+	+	+	+
Liver	0	0	0	0
Meninges	+	+	0	0

COMMENT

Abt and Denenholz [4] reviewed the acute noninfectious forms of nonlipid reticuloendotheliosis in 1936, and feeling this acute form was a clinicopathologic entity separate from Hand-Schüller-Christian disease, gave it the name of Letterer-Siwe disease, with the following characteristics: (1) hepatosplenomegaly; (2) hemorrhagic diathesis, especially petechiae and purpura; (3) generalized lymphadenopathy of a mild to moderate degree; (4) progressive anemia with a usually normal leucocyte count; (5) nonhereditary, nonfamilial occurrence, exclusively in infants and young children, with unknown etiology, fatal prognosis, and variable durations (from a few weeks to several years); (6) localized skeletal tumors; (7) generalized hyperplasia of the reticuloendothelial cells with focal tumorous proliferations, especially in the spleen, liver, lymph nodes, thymus, skin, and bone marrow, often showing a hemorrhagic tendency, but no lipid storage. Wallgren feels basically this is the same granulomatous process which goes on to lipid storage in the macrophages if the disease continues over a long enough period. Rowland's [5] concept of this process as a cholesterol metabolic dysfunction has not been substantiated and is not widely accepted.

This case fills most of the criteria for diagnosis set forth by Abt and Denenholz.[4] Bony tumors were not grossly or roentgenologically present. The microscopic examination disclosed, however, that lesions of mixed type were present in the marrow. The lack of gross or roentgenographically demonstrable change in long bones is similar to the finding in a case reported by Havard, Rather, and Faber.[2a]

4. Abt, A. F., and Denenholz, E.: Letterer-Siwe's Disease: Splenohepatomegaly Associated with Widespread Hyperplasia of Nonlipoid-Storing Macrophages: Discussion of So-Called Reticulo-Endothelioses, Am. J. Dis. Childhood **51**:499-522 (March) 1936.

5. Rowland, R. S.: Xanthomatosis and the Reticuloendothelial System, Arch. Int. Med. **42**:611-674 (Nov.) 1928.

One point not mentioned by Abt and Denenholz [4] as a diagnostic criterion is emphysema. Our patient displayed this in the lung, the mediastinum, and the subcutaneous tissues of the neck and upper anterior chest. Emphysema is mentioned as occurring only in the lungs by Letterer [1] and is mentioned by Imler [6] as being present but limited likewise to the lung as in the cases of Farber [7] and of Lane and Smith.[8] Extension beyond the lungs is mentioned in one of three cases reported by Schafer.[2b] This is a unique finding so far as we can determine.

Massive splenomegaly is almost always a prominent feature of these cases. It can be of remarkable degree. One case is reported to have had an increase of 17.8 times normal.[2b] In our case the degree of enlargement was more nearly that reported by Foot and Olcott,[9] approximately eight times normal. Most of this increase in

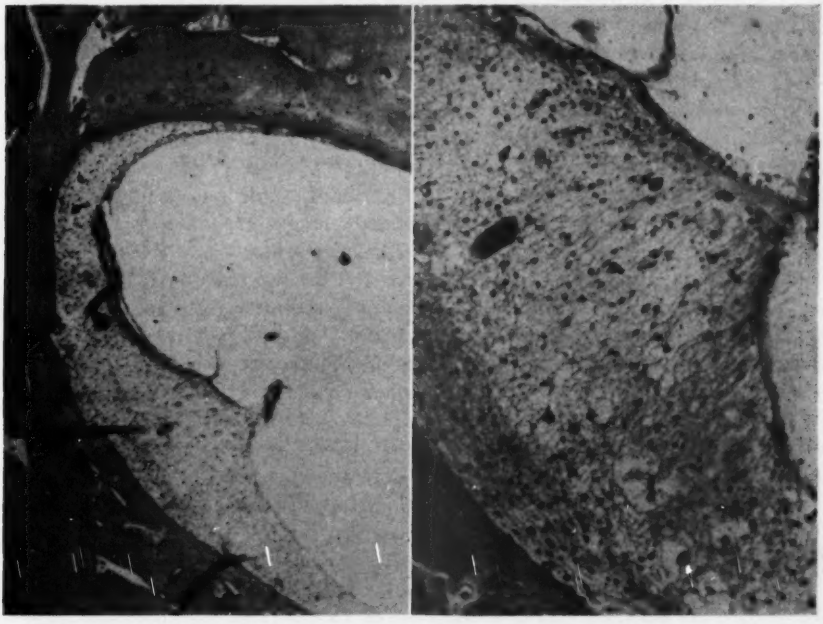

Fig. 3.—Left, one of the cysts in the bone marrow (medium power); right, central portion of the cyst shown at the left, demonstrating the cells lining the cavity and in the wall (high power).

size was observed clinically as having occurred in a period of 10 days. Microscopic section of the spleen shows the splenomegaly to be of the neoplastic type. This

6. Imler, A. E.: Reticulo-Endotheliosis, with Report of 2 Cases, Am. J. Roentgenol. **56**:343-354 (Sept.) 1946.

7. Farber, S., in Pathological Discussion, Case Records of the Massachusetts General Hospital, New England J. Med. **226**:394-395 (March 5) 1942.

8. Lane, C. W., and Smith, M. G.: Cutaneous Manifestations of Chronic (Idiopathic) Lipoidosis (Hand-Schüller-Christian Disease), Arch. Dermat. & Syph. **39**:617-644 (April) 1939.

9. Foot, N. C., and Olcott, C. T.: Report of a Case of Nonlipoid Histiocytosis with Autopsy, Am. J. Path. **10**:81-95 (Jan.) 1934.

indicates the extreme rapidity with which reticuloendothelial cells can proliferate and equals in degree the inflammatory hyperplasia report by Neff and Davis [10] in a case of systemic histoplasmosis.

Most of the reported cases have shown osseous lesions visible by roentgenography. This was not true in our case. The marrow space, however, was not free from involvement microscopically and, in addition to the reticuloendothelial proliferation, exhibited cysts (Fig. 3). These have been reported in only four other cases, as reported by Foot and Olcott.[9] It is felt that the extensive pulmonary lesions were lethal before the marrow changes had progressed to the point of demonstration by roentgenography.

Very recently Dennis and Rosahn,[11] in discussing the mechanism of death in young patients with this disease, have stated that spontaneous pneumothorax was the immediate cause. Our patient, in whom pulmonary cysts and mediastinal and subcutaneous emphysema developed, with the resultant pulmonary atelectasis, died before the development of pneumothorax. It would appear, therefore, that atelectasis, whether mediated by cysts of the lung, mediastinal emphysema, or spontaneous pneumothorax, is the mechanism of death in some of the cases occurring in infants.

Examination of the spinal fluid has been reported, to our knowledge, in but one case; in this case nothing abnormal was found.[12] Three examinations in our case showed a total count of 100 or less, with a relative lymphocytosis. If the spinal fluid had been examined in previously reported cases, it may well have shown similar cell counts, because many of the autopsy summaries contain descriptions of meningeal involvement closely resembling the finding in the case here reported. Whether the mononuclear cells which were reported were actually true lymphocytes or were immature cells of the reticuloendothelial variety is problematical.

Roentgenologists have pointed out the difficulty of distinguishing between the appearance of bone and visceral changes as caused by reticuloendotheliosis and those which result from other commoner etiological agents.[6]

Others have called attention to gradations of pathologic change which relate the clinically benign types to the highly malignant forms.[13]

Otitis media, cholesterolemia, thrombopenia, monocytosis, soft mottling in lung fields on roentgenograms, and early cirrhotic changes in liver have all been mentioned by other authors. These features in our case differ in no important respect from those previously reported.

SUMMARY

A case of fatal reticuloendotheliosis in a 16-month-old infant is presented. Several features, both clinical and pathologic, which have rarely occurred in cases reported by other authors were combined in this case. It is reported in an effort to further delineate this particular disease as a clinical and pathologic entity.

10. Davis, H. V., and Neff, F. C.: Histoplasmosis in Infancy, Am. J. Dis. Child. **71**:171-177 (Feb.) 1946.

11. Dennis, J. W., and Rosahn, P. D.: Primary Reticulo-Endothelial Granulomas, Am. J. Path. **27**:627-654 (July-Aug.) 1951.

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GENERALIZED HERPES SIMPLEX IN AN ELEVEN-DAY-OLD PREMATURE INFANT

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AND
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HERPES simplex is one of the most widely disseminated viruses, and yet the occurrence of herpetic disease in newborn infants seems to be very rare. Scott [1] stated that he had never heard of it, and we have been unable to find any reported cases.[1a]

The present communication is concerned with a unique "experiment of nature." It is a report of an 11-day-old premature infant, one of triplets, who was observed to have blisters on the lips and subsequently a generalized vesicular eruption and meningoencephalitis. She survived with residual neurological defects and chorioretinitis. A virus was recovered from the vesicular fluid and was identified by its pathogenicity, serology, and morphology, as revealed by the electron microscope, as herpes simplex. The patient had neutralizing antibodies for this agent. The two other triplets were sent to different hospitals shortly after birth and thus serve as controls. One infant had no clinical disease, and no antibodies were detected in the serum. The other was also free of obvious herpes infection, but later chorioretinitis and a low titer of herpes-simplex-neutralizing antibody developed.

REPORT OF A CASE

L. H., the third of triplets born at another hospital, was admitted to Lincoln Hospital Premature Nursery Feb. 18, 1950, five hours after birth. Examination at that time revealed a 3-lb-2-oz. (1,417-gm.) baby girl with good color, activity, and fair cry. The liver could be palpated 2 cm. below the costal margin; the spleen tip was barely palpable. The umbilical cord, which was oozing, was retied.

Given the usual care for an infant of her size, she did well at first. Her weight did not drop below 2 lb. 15 oz. (1,332 gm.), and no fluids were required parenterally. Slight jaundice appeared on the third day and lasted for three days. On the 11th day when the weight had increased to 3 lb. 1 oz. (1,389 gm.), two small vesicles were noted on the upper lip. The following day there were many vesicular lesions on both lips, the left side of the face, and the back. These tended to be grouped in clusters. The vesicles varied from 1 to 5 mm. in diameter. They were filled with cloudy fluid and surrounded by a narrow halo of erythema. They dried with crusting and without umbilication. New vesicles continued to appear for 16 days. For the first 10 days they were more numerous on the trunk; thereafter they were practically limited to the extremities. Some vesicles appeared also on the tongue and the palate. On the latter they coalesced to form a tough, greyish, partially detached membrane which had to be removed to prevent obstruction of the pharynx.

From the Division of Bacteriology and the Pediatric Service, Mount Sinai Hospital, and the Department of Pediatrics, Lincoln Hospital and the New York Hospital-Cornell Medical Center.

1. Scott, T. F. M.: Personal communication to the author.

1a. Since this paper was submitted for publication Quilligan and Wilson have reported a fatal case in a newborn infant (Quilligan, J. J., and Wilson, J. L.: Fatal Herpes Simplex Infection in a Newborn Infant, J. Lab. & Clin. Med. **38**:742-746, 1951).

The infant's cry and color were poor during the first few days of the disease. Then, despite the appearance of new lesions, she appeared somewhat better. However, on the 10th day of illness, the 21st day of life, she had a generalized convulsion which lasted several minutes. Lumbar puncture shortly thereafter revealed hazy fluid, with 150 white blood cells, of which 80% were lymphocytes and 20% were polymorphonuclear leucocytes. The sugar in the cerebrospinal fluid was 38 mg. per 100 cc., and the protein was 104 mg. per 100 cc. Bacterial cultures were sterile. Convulsive movements of varying degrees of severity continued intermittently for the next 24 hours in spite of sedation. A second lumbar puncture revealed 180 white cells, with 90% lymphocytes. The amount of sugar in this fluid was not recorded. The convulsions gradually subsided during the next three days, but the baby continued to be irritable and to exhibit moderately severe twitchings when disturbed. She gained no weight. Fifteen days after the onset of the convulsions it was noted that the fontanelle was tense and that sutures were separated about 0.5 cm. There had been no change in the clinical condition. Lumbar puncture at this time yielded deeply xanthochromic fluid under increased pressure. Subdural punctures revealed nothing significant. Lumbar puncture the next day again yielded xanthochromic spinal fluid with a few fresh red blood cells. No further punctures were done. Nevertheless, the fontanelle remained soft, and the head did not increase abnormally in circumference. The hyperirritability of the infant gradually became less marked, and she began to nurse better. The skin cleared without scarring except in areas where there had been extensive sloughing.

Treatment with aureomycin and penicillin was started on the second day of the child's illness. The following day sulfadiazine was added to the therapeutic regimen. Penicillin and sulfadiazine were administered until the 8th day of illness and aureomycin until the 15th day. Chloramphenicol (chloromycetin®) therapy was started on the 10th day and continued until the 20th day. Then a second course of penicillin was begun. However, all antibiotic therapy was discontinued on the 25th day when the xanthochromic spinal fluid was discovered.

The baby's eyes were examined by Dr. Frederick L. Blodi, and the following notes were made: Feb. 23, 1950, "glimpses of normal fundi"; March 2, "fundi normal"; March 30, "only red reflex"; April 13, "normal fundi."

Material from the vesicles and the spinal fluid was taken for virus studies on March 11, 1950, the 11th day of the disease.

The baby was discharged from Lincoln Hospital April 23, 1950, after 65 days in the hospital, having attained a weight of 5 lb. 8 oz. (2,500 gm.). Nevertheless, she still had increased neuromuscular irritability when disturbed.

Her case was followed in the Premature Clinic of the New York Hospital. After six weeks, when 3½ months old, she was admitted to that hospital because of a respiratory infection accompanied with high fever. Shortly after this had subsided, severe diarrhea developed. This required vigorous and prolonged treatment before recovery occurred. Thereafter she remained in the hospital as a boarder for socioeconomic reasons until Sept. 9, 1950, when she was discharged to a foster home. Throughout this hospitalization she was noted to have rigid musculature with occasional tremors, and she tended to lie in opisthotonos. However, there were no further convulsions. Her weight gain was slower than normal.

During the early part of her stay at New York Hospital, the vitreous of each eye was found to be hazy. Subsequently, the ophthalmic picture was found to be that of acute chorioretinitis. Although healing of this lesion occurred during the next two months of observation,[2] extensive scarring and total blindness resulted. Roentgenograms of the skull on several occasions failed to reveal any intracranial calcification.

After six weeks in the foster home, the patient was readmitted to New York Hospital because of an episode of laryngotracheitis with pneumonia. On the fifth day, when the temperature had already returned to normal, a vesicular eruption appeared. There were four clusters of five to eight vesicles each in nonsegmental distribution, two on the right anterior chest wall, one in the left scapular area, and one on the left upper arm. These cleared within 24 hours. There were no new significant findings in any system of the body. The chorioretinitis was unchanged.

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2. The ophthalmoscopic observations were made by Dr. Franklyn P. Bousquet Jr., and Dr. William E. Laupus.

The infant was severely retarded both mentally and physically. She usually lay in opisthotonos, with spastic musculature, and made no attempt to sit up or to roll over.

The fate of each of the other triplets is of interest. N. H., infant 1, also a girl, weighed 2 lb. 13 oz. (1,380 gm.) at birth. She was moved on the first day to New York Hospital, where she had an uneventful two-month stay and went home having attained a weight of 5 lb. 2 oz. (2,340 gm.). At no time did she manifest evidence of systemic infection. However, on the 34th day routine ophthalmoscopic examination revealed for the first time a pathologic condition in the left eye which, during the next three months, was observed to run the course of acute chorioretinitis, healing with a small area of scarring. This infant was readmitted to the hospital, with mild bronchopneumonia, three days after her sibling, when she was 3¼ months old. She too remained for three months as a boarder. She had several episodes of mild diarrhea. Her weight gain and development were normal.

B. H., infant 2, a boy, weighed 3 lb. (1,361 gm.) at birth. He was taken to a third hospital where he is said to have had an uneventful course. He was seen in New York Hospital Premature Clinic several times when he was between 9 and 15 weeks old. Results of examinations, including ophthalmoscopy, were completely normal. When 15½ weeks old, after a few days of

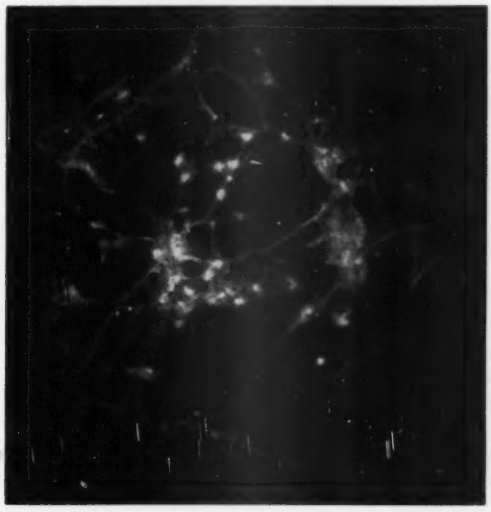

Fig. 1.—Pocks produced by patient's virus on chorioallantoic membrane of chick embryo.

mild respiratory symptoms, he was found dead in his crib the morning of the day his sibling, N. H., was admitted with pneumonia. No autopsy was done.

VIROLOGY

Vesicle and cerebrospinal fluids were obtained from the patient, L. H., on March 11, 1950, on the 11th day of illness. Both fluids were immediately frozen in carbon dioxide snow and brought to Mount Sinai Hospital virology laboratory. On March 14, 1950, the bleb fluid was inoculated onto the chorioallantoic membrane of four 11-day-old embyronated eggs. Three days later each was found to have 20 to 100 pocks suggestive of herpes simplex virus (Fig. 1). An emulsion was prepared and inoculated intracerebrally into white mice and again onto the chorioallantoic membrane of 11-day-old chick embryos. Two of the three mice convulsed on the fourth day, and one of these died. The other was killed and the brain removed. This was reinoculated onto the chorioallantoic membrane of embryonated eggs,

into the brain of more mice, and onto the scarified cornea of a rabbit. Keratoconjunctivitis developed in the rabbit on the third day. The results of the egg and mouse passages were essentially similar to those described above and elsewhere with other strains of herpes simplex.[3] Similar studies carried out with the cerebrospinal fluid failed to reveal the presence of any virus.

Allantoic fluid from one of the later egg passages of the bleb fluid was centrifuged at 10,000 rpm for one hour. The precipitated pellet was resuspended in a small volume of saline solution and examined under the electron microscope by Dr. Gregory Shwartzman. The results are shown in Figure 2. The circular bodies with central depression are very similar to those in pictures of herpes simplex virus shown by others.[4] Their size was calculated to be 166 mμ. This is in agreement with the 165-mμ measurement of the standard herpes febrilis strain of herpes simplex virus prepared in the same fashion in our laboratory.

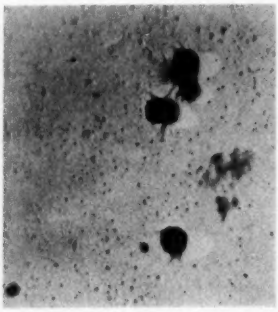

Fig. 2.—Appearance of patient's virus under electron microscope.

Results of Mouse Neutralization Test *

Sera	Virus Dilution					L.D.$_{50}$
	10^{0}	10^{-1}	10^{-2}	10^{-3}	10^{-4}	
Normal rabbit	0/5	0/5	1/5	4/5	4/4	$10^{-3.5}$
Immune rabbit (herpes simplex-J)	...	3/5	5/5	...	...	.....
Convalescent rabbit (patient's virus)	...	4/5	5/5	...	...	.....
Triplet No. 3 (patient), 46th day of life	...	4/5	3/5	...	...	.....
Triplet No. 2, 47th day of life	...	0/5	3/5	...	...	.....
Triplet No. 1, 58th day of life	...	2/5	3/5	...	...	.....

* Numerator of fraction indicates number of mice surviving; denominator indicates number of mice inoculated.

The final identification of the agent as herpes simplex came from neutralization tests employing both the mouse and the chick embryo as test animals. In both tests known herpes simplex immune-rabbit serum neutralized the new virus, as did a homologous immune-rabbit serum (Table).

SEROLOGY

The association of the recovered virus with the patient's illness is shown by the appearance of specific neutralizing antibodies in the serum. The titer of these anti-

3. Florman, A. L., and Trader, F. W.: Comparative Study of Pathogenicity and Antigenicity of Four Strains of Herpes Simplex, J. Immunol. **55**:263-275, 1947.

4. Coriell, L. L.; Rake, G.; Blank, H., and Scott, T. F. M.: Electron Microscopy of Herpes Simplex, J. Bact. **59**:61-68, 1950.

bodies was relatively low as compared with the convalescing rabbit, but was higher than that found in the sera of the patient's siblings. This is shown in the mouse neutralization test summarized in the accompanying table. Similar results were obtained when the neutralization was carried out on the chorioallantoic membrane of chick embryos.

COMMENT

There are several unique features of this case worthy of comment. These include the age of the patient, the possible source of infection, and the residual chorioretinitis.

Anderson and Hamilton [5] in a recent study of the epidemiology of primary herpes simplex infections have indicated that infection is unusual before the age of 8 to 10 months. They suggest that this might reflect the placental transmission of protective antibody. We have carried out experiments which indicate that there is very little difference in the titer of this antibody in maternal and cord blood.[6] On this basis it would be theoretically possible to find this disease in very young children of nonimmune mothers. Presumably this was the situation in our patient, for, although we were unable to obtain blood from the mother, one of the triplets had no protective antibodies. Yet, because of the rarity of this infection in the neonatal period, other predisposing factors may be involved. One of these may be prematurity. The youngest previously reported instance of infection with herpes simplex that we have been able to find is that of Smith, Lennette, and Reames.[7] These authors reported the recovery of herpes virus from the brain of a 4-week-old premature infant who died nine days after the onset of a febrile illness characterized by irritability and convulsive movements.

The usual incubation period for primary herpes simplex gingivostomatitis, the most frequent clinical form of infection with this virus, is generally considered to be three to nine days, although Scott and Steigman [8] stated that it might possibly be longer. In a 1-year-old infant, experimentally infected by Black,[9] fever developed, with erosion of the gums at the site of inoculation, after an incubation period of three days. However, it was not until the ninth day, that the condition was described as typical of gingivostomatitis. Generalized herpes simplex is usually associated with Kaposi's varicelliform eruption.[10] This occurs in children with eczema, and the incubation period is not known. The first signs of herpes in our patient, in whom a generalized infection subsequently developed, were two small vesicles on the upper lip on the 11th day of life.

One can only speculate as to the source of this infection. If the presence of chorioretinitis and low titer of herpes simplex neutralizing antibody in the serum

5. Anderson, S. G., and Hamilton, J.: The Epidemiology of Primary Herpes Simplex Infection, M. J. Australia **1**:308-311, 1949.

6. Florman, A. L.: Unpublished data.

7. Smith, M. G.; Lennette, E. H., and Reames, H. R.: Isolation of the Virus of Herpes Simplex and the Demonstration of Intranuclear Inclusions in a Case of Acute Encephalitis, Am. J. Path. **17**:55-68, 1941.

8. Scott, T. F. M.; Steigman, A. J., and Convey, J. H.: Acute Infectious Gingivostomatitis, J. A. M. A. **117**:999-1005 (Sept. 20) 1941.

9. Black, W. C.: The Etiology of Acute Infectious Gingivostomatitis, J. Pediat. **20**:145-160, 1942.

10. Blattner, R. J.; Heys, F. M., and Harrison, M. L. K.: Etiology of Kaposi's Varicelliform Eruption, J. Pediat. **27**:207-215, 1945.

of one of the other triplets is significant, one might conclude that infection occurred at the time of delivery, since the children were separated shortly thereafter.

The end-result in our patient was healed chorioretinitis and gross cerebral damage without evidence of cerebral calcification. This fits the syndrome recently described by Sabin and Feldman,[11] which they believe differs from congenital toxoplasmosis. No definite etiologic agent has been found, and these authors postulate a multiplicity of causative factors. It would appear from the experience reported here that infection with herpes simplex in the neonatal period might be one of those factors.

SUMMARY

The case history is presented of a premature infant weighing 3 lb. 2 oz., one of triplets, who was discovered on the 11th day of life to have vesicles on the upper lip. A generalized vesicular exanthem, enanthem, and convulsions subsequently developed. The infant recovered but with residual damage to the central nervous system and chorioretinitis.

The virus of herpes simplex was recovered from the skin lesions, and neutralizing antibodies were later found in the serum.

The significance of the youth of this patient, the residual chorioretinitis, and the possible source of infection are discussed.

46 Barberry Lane, Roslyn Heights.

229 E. 79th St. (21).

11. Sabin, A. B., and Feldman, H. A.: Chorioretinopathy Associated with Other Evidence of Cerebral Damage in Childhood: A Syndrome of Unknown Origin Separable from Congenital Toxoplasmosis, Am. J. Dis. Child. **79**:366-368 (Feb.) 1950.

REVERSED ROTATION OF THE BOWEL

Review of the Literature and Report of an Unusual Case

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REVERSED rotation of the mid-gut with resultant retroposition of the transverse colon is probably the rarest of all developmental anomalies of the colon.[1] This unusual condition results from an error in the second stage of rotation of the mid-gut. When the mid-gut returns to the abdominal cavity from the umbilical cord during the 10th week of embryonic life, a 90-degree clockwise rotation occurs (inverted rotation) rather than the normal 270-degree counterclockwise rotation. Hence, the transverse colon passes dorsal to the superior mesenteric artery at the angle of its emergence from the aorta, and the duodenum passes ventral to both.

An unusual case of reversed rotation complicated by partial obstruction of the transverse colon and partial duodenal obstruction will be described, and the 27 previously reported examples[2] of reversed rotation will be discussed.

REPORT OF CASE

S. J. W., a white girl, weighed 8 lb. 10 oz. (3,910 gm.) at birth. She progressed satisfactorily until 11 days of age, when she experienced several episodes of projectile vomiting associated with extremely prominent abdominal peristaltic waves passing from the upper left quadrant to the right of the umbilicus. The vomitus consisted of milk only. These episodes were controlled within 48 hours on a regimen of phenobarbital elixir given before meals and a low-fat milk. Two days later the phenobarbital administration was discontinued without recurrence of vomiting. Several soft stools were passed daily during this period, and at no time was an abdominal mass palpated.

Five weeks later, at 7 weeks of age, an acute vomiting episode, consisting of 20 vomiting spells in a 12-hour period, occurred. Findings were identical to those noted previously, except that at this time the vomitus was bile-green color. She was admitted to Children's Hospital for

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From the Children's Hospital.

1. Truesdale, P. E.: Retroposition of the Transverse Colon: Report of Two Cases, J. A. M. A. **104**:1697 (May 11) 1935.

2. (*a*) Gardner, C. E., Jr., and Hart, D.: Anomalies of Intestinal Rotation as a Cause of Intestinal Obstruction, Arch. Surg. **29**:942 (Dec.) 1934. (*b*) Rose, T. F.: Retroposition of the Transverse Colon Complicated by Ileocaecal Volvulus: A Report of One Case with Recovery, and a Review of the Literature, M. J. Australia **1**:225, 1941. (*c*) McIntosh, R., and Donovan, E. J.: Disturbances of Rotation of the Intestinal Tract, Am. J. Dis. Child **57**:116 (Jan.) 1939. (*d*) King, E. S. J.: Two Uncommon Forms of Intestinal Obstruction Occurring Consecutively: Compound Volvulus and Retroposition of the Transverse Colon, Brit. J. Surg. **24**:817, 1937. (*e*) Murray, N. G.: Retroposition of the Transverse Colon: Report of a Case, New Zealand M. J. **45**:545, 1946.

correction of dehydration and study of the intestinal tract. After two days of parenteral hydration and restoration of electrolytes, she began again to retain her formula. A gastrointestinal fluoroscopic series revealed an obstruction in the third part of the duodenum. The duodenum proximal to the point of stoppage was dilated, and after manual manipulation a small amount of barium passed on into the jejunum. At the end of five hours approximately 90% of the barium had passed the point of obstruction, and at the end of 24 hours all of the barium had been expelled. Fluoroscopic examination of the esophagus and stomach showed them to be normal, and a flat roentgenogram of the abdomen and a roentgenogram of the chest revealed no abnormalities. Urinalyses showed normal results, and a complete blood count gave values within normal limits. At the time of admission she weighed 10 lb. 2 oz. (4,600 gm.), and on discharge at 8 weeks of age she weighed 10 lb. 4½ oz. (4,660 gm.). Although a definite diagnosis of partial intestinal obstruction at the level of the third part of the duodenum was made, it was considered advisable, in view of the excellent weight gains and good physical condition, to observe the patient conservatively until a greater age had been attained.

Two weeks after discharge, cereal was added to her diet, and five days later, at 2½ months of age, she was again admitted to the hospital, because of five episodes of similar projectile vomiting of bile-tinged fluid. Physical examination showed the same results as previously; however, at this time she weighed 12 lb. 6 oz. (5,610 gm.). A blood count disclosed the hemoglobin level to be 10 gm. and the red blood cell count to be 3,600,000. After parenteral correction of the dehydration, a transfusion of 130 cc. of whole blood was administered. A repeat gastrointestinal series confirmed the previously located obstruction; however, at this time approximately 90% of the barium had passed the obstruction at three hours. No vomiting occurred while the patient was in the hospital, and she was discharged 24 hours after admission weighing 12 lb. 5½ oz. (5,600 gm.). Her formula consisted of milk and cereal as previously.

During the next six days she retained all food for periods of 30 to 36 hours, after which she became extremely restless and had projectile vomiting. After vomiting two or three consecutive feedings, she passed an extremely large stool and again began retaining food for 30 to 36 hours. This pattern of vomiting just prior to the passage of a stool persisted until admission to the hospital. At the time of admission, only six days after her previous discharge, she weighed 10 lb. 15 oz. (4,960 gm.). Because of the definite association of large constipated stools with periods of projectile vomiting, a barium enema was performed. The barium was propelled up to the region of the hepatic flexure, where a marked redundancy was noted, i. e., the colon traversed from that point over to the left of the umbilicus and then posteriorly to a point of complete block in the region of the juncture of the first and second thirds of the transverse colon. No barium could be propelled beyond that point, although the hepatic flexure, ascending colon, and cecum were dilated with air and seemed to be in their normal anatomic position. An obstruction in the proximal third of the transverse colon with a redundancy of the distal transverse colon was thus diagnosed. There was also marked redundancy of the sigmoid colon. Twenty-four hours later the gastrointestinal series was repeated, with similar findings of partial obstruction in the third part of the duodenum. In preparation for an exploratory laparotomy, an enema was administered daily, in view of the association of vomiting episodes with the accumulation of large stools. Consequently, no vomiting occurred during the 48 hours of preoperative preparation. An exploratory laparotomy through an upper midline incision was performed (C. S. W.) 48 hours after admission, with the patient under ether-oxygen anesthesia.

The preoperative diagnoses were partial obstruction of the third part of the duodenum and partial obstruction of the proximal third of the transverse colon. A large loop of redundant transverse colon was bound down by adhesions over the third part of the duodenum. After the adhesions were freed, the duodenum was noted to pass anterior to the superior mesenteric artery, whereas the proximal transverse colon passed dorsal to the artery and was situated snugly in the angle of the aorta and the superior mesenteric artery. Pressure from this segment of bowel against the mesenteric vessels produced partial anoxia and cyanosis of the small bowel supplied by the artery. The transverse colon was freed by blunt dissection from the angle of the superior mesenteric artery and aorta and allowed to fall into the pelvis posterior to the small bowel and its mesentery. The entire bowel was on a single mesentery, and no normal fixation was noted. Whole blood was administered preoperatively and during the operative procedure. Streptomycin

and penicillin were administered prophylactically postoperatively, and a stomach tube maintained adequate drainage during the two-day period of postoperative paralytic ileus. Fluid and electrolyte balance was maintained for three days with use of dextrose, protein hydrolysates (amigen®), and Hartmann's solution, after which time a regular diet was instituted, with retention of all formula. Two days postoperatively, the hemoglobin level was 16 gm. and the red cell count was 4,600,000. She was discharged on the sixth postoperative day weighing 12 lb. (5,440 gm.). Physical examination at that time disclosed no abnormalities, and the operative wound was healing well.

Four months after surgery a repeat gastrointestinal series and a barium enema disclosed no obstructive point or any other abnormality. She is now enjoying excellent health at 16 months of age and has had no recurrence of her previous symptoms.

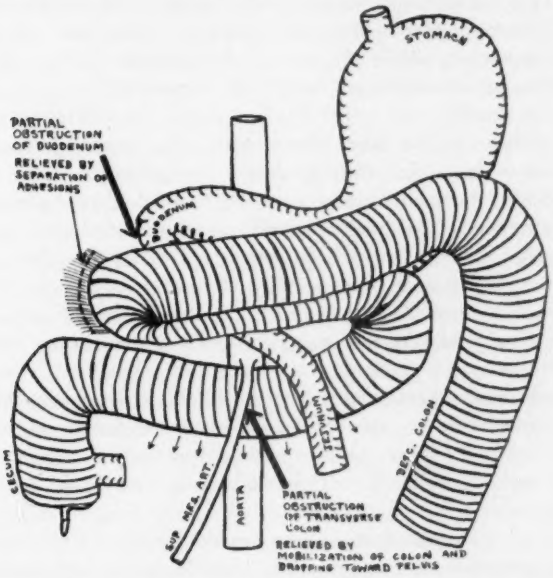

Diagram showing partial obstruction of the duodenum and transverse colon.

COMMENT AND REVIEW OF THE LITERATURE

In a review of the literature from 1916 to 1950, a total of 27 cases of reversed rotation of the gut were found. The majority of these cases were extensively reviewed by Truesdale,[1] Gardner and Hart,[2a] and Rose.[2b] McIntosh and Donovan,[2c] King,[2d] and Murray[2e] discussed additional cases. Sixteen patients were females, and 11 were males. Eight recovered, and 19 died. The complications of reversed rotation fell into four categories, namely, (1) volvulus of the mobile right half of the colon, (2) volvulus about the superior mesenteric root, (3) compression of the transverse colon, and (4) duodenojejunal junction obstruction.

Seventeen patients, with ages ranging from birth to 68 years, presented initial symptoms of acute intestinal obstruction due to volvulus of the mobile right half of the colon (ileocecal volvulus, kink, or torsion).[3] Two diagnoses were made at autopsy, and of the other 15 patients, for whom diagnosis was made at operation,

3. Truesdale.[1] Gardner and Hart.[2a] Rose.[2b] Murray.[2e]

nine died and six recovered. This complication results from retention of the primitive incompletely fixated mesentery of the terminal ileum, cecum, ascending colon, hepatic flexure, and transverse colon proximal to its constriction by the mesenteric root. Gangrene of the involved segment of bowel due to vascular occlusion with or without perforation of the bowel and peritonitis may result.[2a]

Six patients, with ages ranging from 2 weeks to 38 years, were hospitalized for symptoms of acute intestinal obstruction resulting from volvulus of the small bowel and proximal third of the large bowel about the root of the superior mesenteric vessels.[4] Five of the patients were operated on; three of them died, and two recovered. One patient died without operation, and diagnosis was made at autopsy. This complication occurs as the result of inadequate attachment of the common mesenteric root of the entire mid-gut.[2a] Thus, the mid-gut twists its long common mesentery about the axis of the superior mesenteric vessels and produces traction on the mesenteric root.[2c] If uncorrected early, gangrene of the bowel and/or peritonitis may supervene.[1] One of these patients had presented symptoms of chronic partial large bowel obstruction, resulting from constriction of the transverse colon by the overlying superior mesenteric root, for many years.[4a]

In addition to intestinal obstruction due to volvulus, two patients presented symptoms of chronic partial duodenojejunal obstruction beginning at birth and 2 weeks of age respectively.[5] One was operated on for ileocecal volvulus at 15 years of age,[2a] and the other was operated on for volvulus about the superior mesenteric root at 12¾ years of age.[2c] Both recovered. The duodenal obstruction was relieved in each instance by sectioning the peritoneal bands and adhesions that kinked the terminal duodenum.

Four patients, whose ages ranged from 24 to 49 years, presented no symptoms referable to the reversed rotation.[2a] Two died of unrelated conditions, and the diagnoses were made at autopsy, and for two the reversed rotations were discovered coincidentally during surgery for carcinoma of the ovary and carcinoma of the pylorus respectively.

The patient herein reported represents the fifth reported with reversed rotation of the bowel uncomplicated by volvulus and the third with partial duodenal obstruction. The primary factor in the production of the duodenal obstruction was the fecal distention of the overlying redundant distal transverse colon where it was bound down by adhesions over the third part of the duodenum. The partial obstruction of the transverse colon produced symptoms approximately every 36 hours, when the transverse colon passing dorsal to the superior mesenteric root became distended with feces. One might speculate that such distention produced partial compression of the adjacent overlying superior mesenteric vessels with resultant ischemia and anoxemia of the small bowel and proximal third of the large bowel. The paralytic ileus thus produced probably contributed to the acute lower duodenal obstruction. When the large bowel was emptied of feces, all symptoms and signs of intestinal obstruction subsided. This is the first reported case of reversed rotation with such a sequence of events. In summary, distention

4. (*a*) Peignaux, and Fruchard, H.: Troubles digestifs graves chez une jeune fille de quinze ans en relation avec des malformations portant sur le duodénum et le côlon droit, Bull. et mém. Soc. nat. de chir. **56**:335, 1930. (*b*) Truesdale.[1] (*c*) Gardner.[2a] (*d*) McIntosh and Donovan.[2c] (*e*) King.[2d]

5. Gardner and Hart.[2a] McIntosh and Donovan.[2c]

of the transverse colon resulted in (1) obstruction of the lower duodenum, owing to tension of adhesive bands binding the transverse colon over the third part of the duodenum; (2) obstruction of the transverse colon where it passed behind the superior mesenteric root, and (3) paralytic ileus of the entire mid-gut owing to compression of the superior mesenteric vessels.

The diagnosis of reversed rotation of the bowel is a difficult one to establish preoperatively. A partial obstruction at the duodenojejunal junction was diagnosed preoperatively by means of a fluoroscopic gastrointestinal series in one case [2c]; however, in the same case a barium enema disclosed merely a redundancy of the colon. Barium enemas have been of no avail in the few instances in which they have been utilized.[2b] Nevertheless, our case confirms the value of these measures, since a preoperative diagnosis of partial obstruction of the third part of the duodenum and of obstruction of the proximal transverse colon as well as a redundancy of the distal transverse colon was made by fluoroscopic gastrointestinal series and a barium enema.

Varied forms of surgical management were employed in correcting the volvuli involving the right half of the colon and the mid-gut. In all cases surgery was considered imperative for relief of the acute obstruction, and in all the first step was untwisting, or detorsion, of the volvulus. From that point on, various forms of handling were employed. Four cases involved merely reducing the volvulus and closing the abdomen. All of the patients died. Resection of nonviable bowel and primary anastomosis were accomplished in three cases; i. e., (1) resection of cecum with ileotransverse colostomy, (2) right hemicolectomy with ileotransverse colostomy, and (3) resection of cecum and ascending colon with ileotransverse colostomy. All three patients died. It was recommended by the authors that future resections should involve exteriorizing the volvulus over a clamp. Anastomoses bypassing the partial obstruction to the transverse colon were performed in two cases. In one the cecum was anastomosed to the sigmoid colon,[2d] and in the other a lateral transverse colocolostomy was accomplished.[2a] Both patients recovered. Cecostomies were utilized in five cases in order to fixate the cecum in its normal position in the right iliac fossa, thus preventing a future recurrence of volvulus. This procedure also served to correct the torsion of the bowel and mesentery and to relieve the constriction in the transverse colon.[6] Three of these patients recovered, and two died. Cecopexy, or fixation of the cecum in the right iliac fossa, was performed in three cases.[7] All patients recovered.

Chronic partial duodenojejunal obstruction was handled by sectioning the peritoneal bands maintaining the sharp angulation in one case [2c] and by sectioning bands about an abnormally fixated duodenojejunal junction and utilizing an anterior gastrojejunostomy in the other case.[4a] In both the obstructions were relieved, and uneventful recovery followed.

In the one case in which chronic symptoms of partial obstructive constriction of the transverse colon were presented,[1] the tunnel through which the transverse colon passed (formed by the juncture of the superior mesenteric root and the aorta) was manually dilated, with resultant subsidence of symptoms. No relief of the constriction of the retroposed colon was necessary in the other cases.

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6. Truesdale.[1] Rose.[2b] King.[2d] Murray.[2e]

7. Gardner.[2a] Rose.[2b] McIntosh and Donovan.[2c]

The operative procedure in our case differed from those reported in that the adhesive bands binding the redundant distal transverse colon over the terminal duodenum were sectioned and the proximal transverse colon mobilized from its position in the angle of the aorta and origin of the superior mesenteric artery and dropped into the pelvis, still remaining, however, behind the small bowel as before. Thus, the duodenal obstruction, pressure on the superior mesenteric vessels, and obstruction of the transverse colon were relieved.

*Results in Twenty-Seven Previously Reported Cases of Reversed Rotation of the Mid-Gut**

	Symptomatic Cases (Acute Intestinal Obstruction)				
	Obstruction Due to Ileocecal Volvulus		Obstruction Due to Volvulus About Superior Mesenteric Root		Asymptomatic Cases, No. Patients Died
	No. Patients Recovered	No. Patients Died	No. Patients Recovered	No. Patients Died	
Resection and anastomosis	..	3	..	..	..
Anastomosis only	1	..	1	..	..
Cecostomy	3	2	..	..	..
Cecopexy	2	..	1	..	..
Reduction only	..	1	..	3	..
Diagnosis missed at surgery	..	3	..	..	..
Diagnosis at autopsy; no surgery	..	2	..	1	..
Patient died of unrelated conditions; diagnosis at autopsy	..	..	..	..	2
Diagnosis during surgery for unrelated carcinoma	..	..	..	..	2

* The references for these cases are given in Footnotes 1 and 2.

SUMMARY

Reversed rotation of the bowel is extremely rare in all age groups. Volvulus is the major complicating factor.

An unusual case complicated by partial obstruction of the transverse colon and partial obstruction of the duodenum is reported. This represents the third patient with partial duodenal obstruction and the fifth without complicating volvulus.

The 27 previously reported cases are reviewed.

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MALIGNANT NEPHROSCLEROSIS WITHOUT RETINAL EDEMA IN A CHILD

Long-Term Observations and Renal Function Studies

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S. A. KAPLAN, M.D.
AND
W. A. BRODSKY, M.D.
CINCINNATI

ARTERIOLAR nephrosclerosis is the term applied to involvement of the renal area by hypertensive vascular disease. The benign form is characterized by sclerosis of small arteries and arterioles, while the malignant form has in addition necrotizing vascular lesions which involve particularly the vasa afferentia supplying the glomeruli.[1] Malignant nephrosclerosis is the typical renal lesion of malignant hypertension, although it is by no means always present in that disease [2] and may also occur in far-advanced glomerulonephritis [3] and pyelonephritis.[4]

Malignant hypertension is a clinical condition characterized by persistent hypertension, a progressive downward course, and distinctive changes in the retina (edema of the disc or retina, hemorrhages, and exudates). Renal failure is a late or terminal event.[5] The constancy of the typical retinal changes has been emphasized in studies of the disease both in adults [6] and in children.[7]

Excluding cases of far-advanced chronic glomerulonephritis or pyelonephritis, we are aware of only two cases [8] in which the fully developed histological picture of malignant nephrosclerosis has been described in children. Both of these children had the clinical picture of malignant hypertension.

The case to be presented is of interest because of the history of a nephrotic episode of glomerulonephritis which healed without affecting kidney function. After this, persistent hypertension was present, and, shortly before death, retinal exudates, but never edema of the disc or retina. Necropsy revealed only occasional glomeruli which showed evidence of the healed glomerulonephritis, while

1. Anderson, W. A. D.: Pathology, St. Louis, C. V. Mosby Company, 1948.

2. (*a*) Keith, N. M.; Wagener, H. P., and Kernohan, J. W.: The Syndrome of Malignant Hypertension, Arch. Int. Med. **41**:141 (Feb.) 1928. (*b*) Murphy, F. D., and Grill, J.: So-Called Malignant Hypertension: A Clinical and Morphologic Study, ibid. **46**:75 (July) 1930.

3. Shapiro, P. F.: Malignant Nephrosclerosis: Pathogenesis, Arch. Int. Med. **48**:199 (Aug.) 1931.

4. Weiss, S., and Parker, F., Jr.: Pyelonephritis: Its Relation to Vascular Lesions and to Arterial Hypertension, Medicine **18**:221, 1939.

5. (*a*) Keith, Wagener, and Kernohan.[2a] (*b*) Ellis, A.: Malignant Hypertension, Lancet **1**:977, 1938.

6. Murphy and Grill.[2b] Wagener, H. P., and Keith, N. M.: Diffuse Arteriolar Disease with Hypertension and the Associated Retinal Lesions, Medicine **18**:317, 1939.

7. Court, D.: Malignant Hypertension in Childhood, Arch. Dis. Childhood **16**:132, 1941.

8. Court.[7] Klemperer, P., and Otani, S.: "Malignant Nephrosclerosis" (Fahr), Arch. Path. **11**:60 (Jan.) 1931.

typical lesions of malignant nephrosclerosis were present. The case is also of interest because of the opportunity it afforded of following a child with malignant nephrosclerosis at regular intervals from the onset of hypertension until death. Once during the early period of the disease and again during the final month of illness renal function could be assessed in a fairly comprehensive manner.

REPORT OF A CASE

L. S. was born Sept. 20, 1938. His medical history during infancy and early childhood was not remarkable, and his family history contained no evidence of hypertension or renal disease.

In December, 1943, at the age of 5 3/12 years, he was admitted to a hospital because of edema, and the diagnosis of "nephrotic episode of chronic glomerulonephritis" was made. Urinalysis revealed albumin (3+), frequent hyaline and granular casts, but no red or white blood cells. The sedimentation rate was elevated, and the blood pressure was 116/82 mm. Hg (table). The edema and albuminuria cleared rapidly, and in 10 days the patient was discharged.

During the ensuing four months he remained asymptomatic, although at times albuminuria and microscopic hematuria were present. The serum cholesterol level was markedly elevated on several occasions, and the serum albumin was low.

In July, 1944, he was readmitted to the same hospital because of recurrence of edema and ascites. At that time his blood pressure was 120/70 mm. Hg. After October, 1944, no more red blood cells were observed in the urine, although albuminuria and casts were frequently found.

The pertinent laboratory findings during the period from October, 1944, to August, 1946, are recorded in the table. The child contracted varicella in February, 1945, and mumps in March, 1945, and made an uneventful recovery from both diseases.

He was seen in the nephritic clinic of the Children's Hospital of Cincinnati for the first time in August, 1946, at the age of 8 years, with a history of two episodes of edema during the previous six months. He was well developed, well nourished, and free of edema. His blood pressure was 125/75 mm. Hg. The rest of the physical examination, including funduscopy, revealed no abnormalities. Hemoglobin was 13.4 gm. per 100 cc. Urine showed albumin (2+). The sedimentation rate was 8 mm. an hour.

The patient thereafter remained free from edema and was followed in the nephritic clinic over the next four and one-half years at intervals of one to three months. Representative laboratory data may be seen in the table. Apart from polydipsia and urinary frequency, he remained symptom-free. Prophylactic sulfadiazine medication, which had been begun during the winter of 1945, was continued in a dosage of 0.5 gm. twice daily until April, 1947, when it was discontinued.

Between November, 1946, and October, 1950, repeated urinalyses revealed no more than a trace of albumin and, on two occasions, a few red blood cells. Concentrating ability, serum nonprotein nitrogen, and urea clearances were normal on several occasions.

The child's blood pressure fluctuated but always was somewhat above the levels ordinarily observed at his age. However, in November, 1947, it rose to 140/100 mm. Hg and from that time on remained definitely elevated, increasing gradually and steadily up to the time of death.

In January, 1948, an electrocardiogram and an intravenous pyelogram were made and did not show any abnormality.

Repeated examinations revealed the eye grounds to be normal until July, 1948, when narrowing of the arterioles was first noted. In November, 1948, the supracardiac shadows were found to be abnormally prominent by roentgenography, suggesting aortic dilatation and perhaps some aortic elongation, especially of the ascending aorta.

In August, 1948, the patient was admitted to the hospital for nine days. Funduscopic examination at this time showed spasm of the retinal arterioles (vein to artery ratio: 4 to 2) but no tortuosity of the vessels, no arteriovenous nicking, no exudates or hemorrhages.

The psychiatric consultant did not feel that in this instance the hypertension would be modified to a significant degree by psychotherapy.

A piperoxan hydrochloride test revealed no evidence of pheochromocytoma.

A loading test with hypertonic mannitol done during the hydropenic state [9] showed ability to concentrate to 1,147 milliosmoles per liter in the preloading period and a normal relationship

9. Rapoport, S.; Brodsky, W. A.; West, C. D., and Mackler, B.: Urinary Flow and Excretion of Solutes During Osmotic Diuresis in Hydropenic Man, Am. J. Physiol. **156**:433, 1949.

Representative Laboratory Data

Date		Blood Pressure, Mm. Hg	Urine *			Urea Clearance, % Average Normal	Sed. Rate, Mm./Hr.	NPN, Mg./100 Cc.	Serum Albumin, Gm./100 Cc.	Serum Globulin, Gm./100 Cc.	Serum Cholesterol, Mg./100 Cc.
			Specific Gravity	RBC	Albumin						
1943	Dec.	116/82	.....	None	3+	..	38	32	1.77	2.21	266
1944	March-April	115/75	.....	Occ.	2+	..	36	..	2.90	2.41	500
1944	Oct.	.....	.....	.....	2+	..	..	..	1.94	2.47	571
1945	March-April	.....	.....	.....	3+	..	..	34	3.32	1.98	...
1946	July-Aug.	132/100	1.018	Occ.	2+	..	8	32	....	....	223
1946	Nov.	122/80	1.024	None	Tr.	..	..	34	5.40	1.20	114
1947	April	105/65	.....	Occ.	Tr.	85	..	..	....	....	...
1947	Nov.	140/100	1.018	None	Tr.	..	5	..	....	....	...
1948	Jan.	140/100	1.015	None	0	110	..	..	....	....	...
1948	March	150/100	1.018	None	0	..	..	44	3.30	3.00	180
1948	Aug.-Sept.	162/118	1.024	None	Tr.	97	..	36	4.70	2.30	142
1948	Dec.	186/140	1.022	.....	...	107	..	..	....	....	...
1949	Jan.	180/120	1.016	None	Tr.	..	5	24	....	....	...
1949	March-April	192/140	1.021	None	Tr.	..	..	..	....	....	...
1949	July-Aug.	178/140	.....	Occ.	0	..	..	28	....	....	...
1949	Dec.	178/140	.....	.....	...	62	..	40	....	....	...
1950	Feb.-March	180/130	1.020	None	Tr.	80	..	36	....	....	...
1950	April	184/140	.....	None	Tr.	100	..	35	....	....	...
1950	Aug.	195/165	.....	.....	...	..	6	28	4.40	1.70	...
1950	Oct.	204/170	.....	None	2+	..	..	..	5.00	2.00	...
1950	Dec.	200/160	.....	Occ.	1+	..	..	39	....	....	...
1951	Jan. 25	250/190	1.008	None	4+	..	..	45	....	....	...
1951	Jan. 31	230/160	1.022	None	4+	43	..	38	....	....	...
1951	Feb. 11	200/170	.....	7-15 †	3+	41	..	30	....	....	...
1951	Feb. 23	.....	.....	None	4+	..	..	82	....	....	...

* Occ. indicates occasional; tr., trace.
† Per high power field.

between flow and load after loading. Glomerular filtration rate (mannitol clearance) and renal plasma flow (paraaminohippurate clearance) were within normal limits. Calculated renal osmotic work fell slightly short of normal values.

By December, 1948, the blood pressure had gradually risen to 186/140 mm. Hg, and he was admitted for the second time to the Children's Hospital of Cincinnati, for the purpose of establishing whether sympathectomy could be of benefit for his hypertension. During seven days of hospitalization, studies were performed by members of the "Hypertensive Laboratory" of the Cincinnati General Hospital; these studies showed a slight rise of blood pressure (from 170/130 to 188/148 mm. Hg) in response to the cold pressor test. Three cubic centimeters of tetraethylammonium chloride injected intravenously produced no significant fall in blood pressure but did somewhat decrease the blood pressure rise obtained with the cold pressor test.

Other studies performed in February, 1949, demonstrated that the lowest level of blood pressure obtained after spinal anesthesia to a level of the fourth thoracic segment was 160/130 mm. Hg. Further paralysis of sympathetic tone with tetraethylammonium chloride produced only slight additional fall to 150/110 mm. Hg. It was concluded that neurogenic tone played no significant role in the maintenance of the hypertension and that an operative procedure was not indicated.

His weight reached a maximum of 36 kg. in April, 1950, and during the remaining 10 months of life no additional weight gain occurred. At the end of the school term in June he failed to be promoted to the sixth grade of school.

In August, 1950, his blood pressure was 195/165 mm. Hg, and in October, 210/165, where it remained until his final admission. In November, he complained of headaches and began to lose weight. The headaches gradually increased in frequency and severity and became associated with dizziness and fatigue. Early in January, 1951, he began to experience nausea and vomiting.

On Jan. 25, because of loss of weight and frequent severe headaches, he was admitted to the hospital for the fourth time. Physical examination revealed a well-developed, moderately well-nourished, 12-year-old white boy, 146 cm. in height and weighing 31.5 kg. There was no peripheral edema, and he did not seem seriously ill. His blood pressure was 250/190 mm. Hg. Eyegrounds showed arteriolar constriction and a few small exudates near the disc margin on one side. The rest of the physical examination revealed no abnormalities.

The admission laboratory studies revealed a hemoglobin level of 13.1 gm. per 100 cc. and 11,300 white blood cells. The pH of the urine was 7.5, and the specific gravity was 1.008. The albumin reaction was 4+. White blood cells were not present, but a few red blood cells were found. Serum chemical determinations were as follows: pH 7.54, carbon dioxide 36.4 mEq. per liter, carbon dioxide tension 45 mm. Hg, chloride 85.6 mEq. per liter, nonprotein nitrogen 45 mg. per 100 cc., calcium 9.1 mg. per 100 cc., phosphorus 4.3 mg. per 100 cc. During a urine concentration test the specific gravity of the urine rose to 1.022. Roentgenography demonstrated no change in the cardiac and supracardiac shadows from those previously reported. However, unipolar electrocardiography now showed evidence of left ventricular hypertrophy.

The patient's hospital course was punctuated by persistent vomiting, frequent severe frontal headaches, crampy abdominal pain, anorexia, and nausea. The urine albumin reaction continued to be 4+, but red blood cells were observed on only a few occasions. The serum pH varied between 7.51 and 7.62, the carbon dioxide content between 33 and 38 mEq. per liter, the carbon dioxide tension between 39 and 45 mm. Hg, and serum chloride between 81.6 and 90.4 mEq. per liter. On Feb. 2 the serum sodium was 146 mEq., and the serum potassium, 2.9 mEq. per liter. Serum calcium on two occasions was 9.1 and 9.4 mg. per 100 cc. respectively, and serum phosphorus varied between 2.5 and 4.3 mg. per 100 cc.

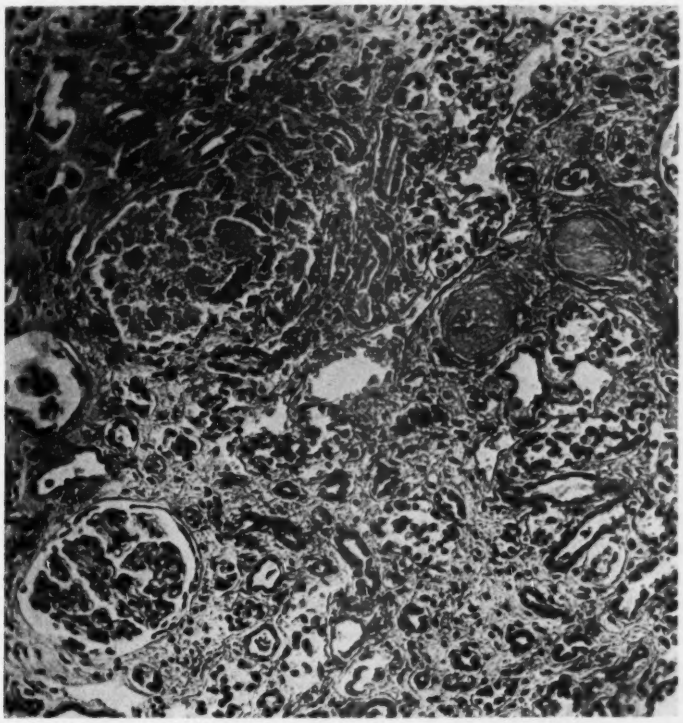
Because of the persistently low serum chloride levels, large doses of sodium chloride were administered orally. On Feb. 4 he was given a total of 15 gm. of sodium chloride with no increase in vomiting. His 24-hour urine output rose from 1,834 cc. on the previous day to 3,080 cc. on the day of the test, and 238 mEq. of chloride (about 14 gm. of sodium chloride) was excreted. The serum chloride level rose from 86.8 mEq. before administration of sodium chloride to 90.0 mEq. on the following morning.

Preliminary renal function studies showed moderately severe impairment: urea clearance was 43% of average normal function; phenolsulfonphthalein was excreted to 45% in one hour. On Feb. 11 more complete studies were performed. The average glomerular filtration rate (inulin clearance) was 60 cc. a minute (normal range: 110 to 130 cc.). Renal plasma flow (para-

aminohippurate clearance) was 165 cc. a minute (normal range: 550 to 650 cc.). Filtration fraction was 37%. Urea clearance averaged 41 cc. a minute (normal range: 60 to 80 cc.). The maximum tubular excretory capacity for paraaminohippurate was 27 mg. a minute (normal range: 50 to 70 mg.).

A muscle biopsy was performed on Feb. 6 to rule out periarteritis nodosa, and no inflammatory reaction was found on microscopic examination.

Ecchymotic and purpuric spots were noted on the patient's legs on two occasions, and his gums were observed to bleed when he brushed his teeth. On Feb. 4 he passed grossly bloody liquid stools and the hemoglobin level fell to 6.9 gm. per 100 cc. He was given transfusions of blood twice, after which the hemoglobin rose to 9.7 gm. per 100 cc., red blood cell count to



Section of the renal cortex, demonstrating arteriolar necrosis with obliteration or marked narrowing of arteriolar lumina, interstitial fibrosis, slight interstitial lymphocytic infiltration, and tubular atrophy (low power).

3,400,000. No further bloody stools were noted until Feb. 15, although microscopic hematuria was consistently present. Blood thrombocyte (platelet) counts varied between 60,000 and 130,000, and the prothrombin time was 50% of normal. The bleeding time was 2½ minutes, the coagulation time (Lee and White) 10½ minutes, and clot retraction was complete in 7 hours.

On Feb. 15 the patient showed a marked change for the worse. He again began to pass large, bloody stools, and transfusions, averaging 500 cc. of whole blood daily, failed to raise his hemoglobin level above 8.5 gm. per 100 cc. Vomiting became more frequent, and beginning on Feb. 18 frank blood was occasionally vomited. On that day he also began to have a fever.

His abdomen remained soft and was not tender until Feb. 21, when distention and tenderness were first noted. No abdominal masses were palpable. His temperature spiked as high as 41.5 C. (106.7 F.); his pulse was weak, and his respirations were at times irregular.

On Feb. 22, the spiking fever continued and abdominal distention and tenderness increased. Fecal vomiting occurred, and a hard abdominal mass was palpated in the left lower quadrant. Exploratory laparotomy was performed and revealed clear, brownish-red peritoneal fluid of excessive amount. Multiple dilated loops of small bowel were seen proximal to an obstruction of the lower jejunum or upper ileum caused by a hard, purplish-white mass of bowel which was thought to represent an old infarct with severe serosal reaction and adherent omentum. The entire small bowel distal to the obstruction was collapsed, purplish in color, and contained multiple small, hard, white areas which were thought to represent additional old infarcts.

The patient's condition was considered to be too critical to warrant any further operative procedure. He became steadily weaker and died on Feb. 24.

A necropsy was performed by Dr. Ralph J. Johansmann: The pertinent gross findings were limited to the cardiovascular, alimentary, and urinary systems. The heart was hypertrophied and moderately dilated, weighing 265 gm. (approximately twice normal weight). Small yellow atheromatous plaques were present in the anterior leaf of the mitral valve, in the intima of the coronary arteries, and in the aorta and the celiac, mesenteric and iliac arteries.

The abdominal cavity contained about 50 cc. of bloody fluid, and the loops of bowel were adherent by fibrinous adhesions. Multiple segments of the small intestine were gangrenous, and the lumen of the bowel was filled with blood.

The kidneys were of average weight. The surfaces were finely granular, and the cortex was mottled yellowish-tan and reddish-brown and speckled with an occasional petechial hemorrhage.

Microscopic examination revealed generalized hyperplastic arteriolosclerosis. In the small arteries there were superimposed arteriolar necrosis and thrombosis and an occasional acute lesion of periarteritis nodosa. The kidneys showed signs of healed glomerulonephritis, as evidenced by an occasional hyalinized glomerulus, hyalinized epithelial crescent, or old adhesion between glomerulus and capsule. On the other hand, marked hyperplastic arteriolosclerosis with arteriolar necrosis, glomerular necrosis, and miliary cortical infarcts were found. Focal zones of tubular and interstitial inflammation and fibrosis alternated with zones of tubular dilatation (figure).

Permission to open the calvaria for study of the central nervous system was not obtained.

COMMENT

The patient presented a rather typical nephrotic episode of glomerulonephritis beginning in December, 1943, when he was 5 3/12 years old. Peripheral edema, albuminuria, elevated erythrocyte sedimentation rate, elevated serum cholesterol, and reduced serum albumin recurred intermittently almost to the time of his eighth birthday (August, 1946). The edema then cleared, and he remained asymptomatic until the age of 11 7/12 years (April, 1950). During this period no more than a trace of albumin was found in the urine, and hematuria was absent except on two occasions, when a few red blood cells were found. Concentration of the urine above a specific gravity of 1.020 was demonstrated repeatedly. The sedimentation rate was normal, as were serum nonprotein nitrogen and urea clearance. Results of more comprehensive renal function studies performed in August, 1948, were normal apart from a defect in performance of osmotic work.

The blood pressure was definitely and progressively elevated beginning November, 1947. Spasm of the retinal arterioles was first noted in July, 1948.

Weight gain ceased in April, 1950, and albuminuria began in October. Headaches, the first symptom directly referable to his hypertension, began in November. In January, 1951, vomiting began and for the first time retinal exudates were present. Renal studies demonstrated markedly impaired glomerular and tubular function.

Terminally his course was marked by severe headaches, abdominal pain and vomiting, gross melena, and coma. During the last few days of life albuminuria

and hematuria occurred, and the serum nonprotein nitrogen level rose to 82 mg. per 100 cc. Death was due to intestinal obstruction caused by mesenteric thrombosis with multiple infarcts of the small bowel.

A vascular accident of the gastrointestinal tract was also the cause of death in the case of malignant nephrosclerosis in a child reported by Court in 1941. He could find no similar case in the literature.

The patient's persistent vomiting was perhaps sufficient to account for the metabolic alkalosis which was present during his final admission to the hospital, but it is significant that a "chloride-losing state" was demonstrated at that time. When large amounts of sodium chloride were administered, more than 90% was recovered in the urine, and the low serum chloride value persisted.

In the presence of ecchymotic and purpuric manifestations as well as hematuria, melena, hematemesis, and death as the result of a vascular accident, persistently low thrombocyte counts and a prolonged prothrombin time are of interest. While bleeding phenomena are common in cases of rapidly progressive hypertension,[10] thrombocyte counts and prothrombin times have rarely been reported, and then have generally been normal.[11]

The absence of edema of the disc or retina even at a time that renal function had become seriously impaired excludes the diagnosis of malignant hypertension.[6] On clinical grounds the case can be classified only as a case of generalized vascular disease with persistent and progressive hypertension in the absence of edema of the disc or retina and with adequate renal function until the terminal phase of illness.

On pathological grounds a definite diagnosis can be made, since the classic histological picture of malignant nephrosclerosis was present. It is notable that there was no evidence of pyelonephritis and only minor evidence of glomerulonephritic scarring.

The role played by the initial episode of glomerulonephritis in the subsequent development of malignant nephrosclerosis cannot be determined, although persistent hypertension has been reported to follow acute glomerulonephritis even when the renal lesion had apparently healed.[12]

SUMMARY

A case of malignant nephrosclerosis in a child in the absence of papilledema is reported. There was an initial episode of a nephrotic stage of glomerulonephritis which was followed by apparently complete recovery. Subsequently hypertension occurred and blood pressure became progressively elevated. Renal failure was a terminal event, and death resulted from a vascular accident of the gastrointestinal tract. Necropsy findings are presented.[13]

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10. (*a*) Keith, Wagener, and Kernohan.[2a] (*b*) Court.[7] (*c*) Guild, H. G.; Kindell, F. B., and Gibson, T. A.: Arteriolosclerosis in Childhood, with a Report of 2 Cases, Bull. Johns Hopkins Hosp. **62**:159, 1938.

11. Court.[7] Guild, Kindell, and Gibson.[10c]

12. Kimmelstiel, P., and Wilson, C.: Benign and Malignant Hypertension and Nephrosclerosis: A Clinical and Pathological Study, Am. J. Path. **12**:45, 1936.

13. While this paper was in preparation a report of two additional cases of malignant nephrosclerosis in children appeared (Zuelzer, W. W.; Charles, S.; Kurnetz, R.; Newton, W. A., Jr., and Fallon, R.: Circulatory Diseases of the Kidneys in Infancy and Childhood, A. M. A. Am. J. Dis. Child. **81**:1 [Jan.] 1951).

PATHOGENESIS OF INTRATHORACIC GASTROGENIC CYSTS

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CONGENITAL anomalies of the vertebrae occurring together with intrathoracic gastrogenic cysts were studied, in an attempt to clarify the pathogenesis of these cysts.

REPORT OF A CASE

A 4½-month-old boy was brought to the Wilhelmina Children's Hospital on June 12, 1948. He had been born spontaneously at full term, with a birth weight of 3,100 gm., and had been breast-fed exclusively. The development had been normal, the infant weighing approximately 5,800 gm. at the time of admission. There had been no previous illnesses. The father and mother were 23 and 20 years old respectively; one other child was 20 months. The father had pulmonary trouble accompanied with a cough. This condition had been present for the previous five months.

From information received from the relevant clinic it became known that he had pulmonary tuberculosis and that his sputum gave a positive reaction for tubercle bacilli. Upon admission it was learned that the baby had been coughing for months and during the previous four weeks had been having fever intermittently. Because of the father's condition a fluoroscopic examination was done on the child in the outpatient clinic, and a shadow was found in the left half of the thorax. The child was then admitted to the hospital.

Examination revealed a well-nourished child with a weight of 5,830 gm. Routine examination of the head, neck, abdomen, genitals, and extremities revealed nothing abnormal. On percussion and auscultation the heart was found to be normal. Percussion and auscultation of the right lung also showed nothing abnormal. There was dullness on percussion over the left lung, and breath sounds could hardly be heard. The temperature was normal. Examination of blood, urine, and feces gave normal results. Pirquet and Mantoux reactions were negative. Repeated bacteriological examinations of the gastric washings were negative for tubercle bacilli.

On fluoroscopic examination (June 17) the entire left hemithorax was found to be shadowed. It was impossible to determine in which part of the left hemithorax the mass was localized. A posteroanterior roentgenogram also showed a massive shadow of the entire left thorax (Fig. 1). In deepest inspiration the roentgenogram presented a homogeneous shadow with a semicircular contour (Fig. 2). Below the shadow there was air-containing tissue. This roentgenogram strongly resembled those found in ganglioneuroma of the sympathetic nerve, in which condition the shadow is also homogeneous and of a semicircular shape. These tumors are always situated in the posterior mediastinum. Transverse roentgenograms did not give a definite picture of the contour.

The bronchogram showed a normal bronchial tree in the right half of the thorax; on the left the iodized oil passed along the shadow and filled a few bronchial ramifications below it. It was impossible to determine which bronchi were filled. The esophagus was considerably displaced to the right (Fig. 2). On June 30 roentgenograms were made in an oblique projection for more precise localization. The results are shown in Figure 3. These roentgenograms showed that the mass causing the shadow was situated in the posterior part of the left hemithorax. During the patient's first month in the hospital, the cough grew worse and swallowing became more difficult. On July 20 a diagnostic puncture was done in the sixth intercostal space in the posterior axillary line. After perforating a rather firm wall, the syringe filled immediately with a slightly

From the University Pediatric Department (Head, Prof. Dr. A. ten Bokkel Huinink), Wilhelmina Children's Hospital.

yellow, almost clear liquid; 70 ml. of this fluid was withdrawn. After this roentgenograms were made. A cyst the size of an egg had become visible in the posterior upper part of the left thorax, close to the spine (Figs. 4 and 5).

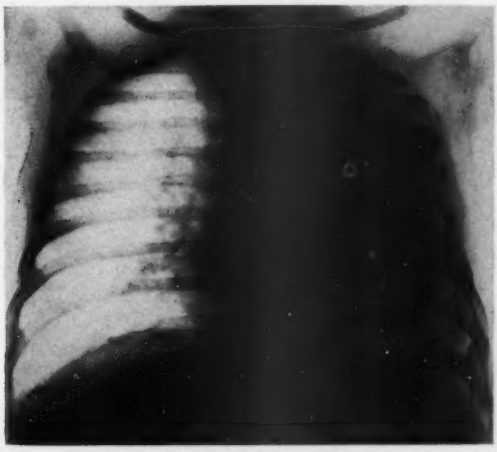

Fig. 1.—Shadow of left hemithorax.

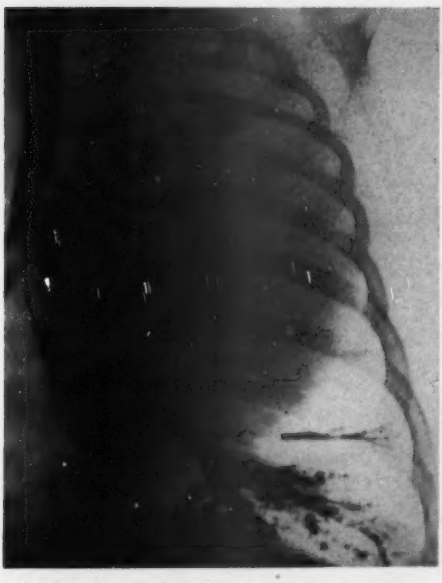

Fig. 2.—In deep inspiration the shadow has a semicircular contour. Displacement of the esophagus to the right and compression of the left bronchial tree are seen.

The following diagnostic possibilities were considered: (1) intrathoracic meningocele: in which case the calcium level of the fluid should be 50% of that in the blood; (2) intrathoracic cyst lined by gastric mucosa: in which case a proteolytic ferment could be demonstrable in the

Fig. 3.—Diagram of results seen on roentgenograms made in the oblique position.

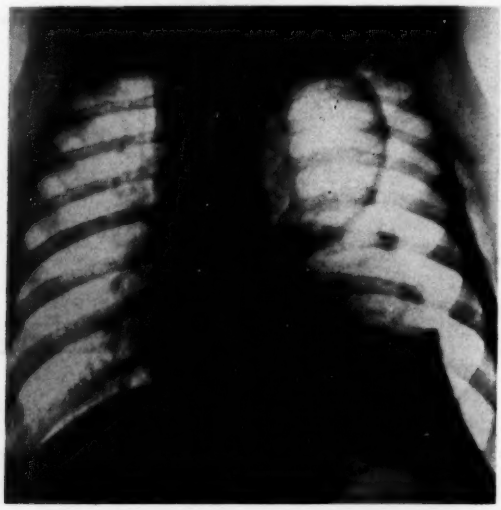

Fig. 4.—Posteroanterior projection after puncture, showing the position of the cyst in the left hemithorax.

Fig. 5.—Lateral projection after puncture, showing the position of the cyst in the posterior mediastinum.

fluid, and (3) Echinococcus cyst: in which case parts of the parasite should then probably be found in the fluid. In regard to the first, the calcium content of the fluid was 7.6 mg. per 100 cc., 9.2 mg. per 100 cc. in the blood and 4.9 mg. per 100 cc. in the cerebrospinal fluid. In regard to the second, a proteolytic ferment comparable in action to about 16 units of pepsin was found in the punctate (Fuld's method). This ferment could not be demonstrated in the cerebrospinal fluid. The pH was 7.4. In regard to the third, Echinococcus hooklets were not found. The Casoni and Weinberg reactions were negative. The fluid was sterile. On the basis of these findings, the conclusion was reached that secreting gastric mucosa was present in the wall of the cyst.

After the puncture, the cough disappeared and swallowing became easier. A few hours later the cyst had again filled with fluid. The amount increased daily, and after 14 days the symptoms had reappeared. Operative removal of the cyst seemed therefore indicated, the more so as it is known that a perforating peptic ulcer may develop, and the contents of the cyst then exerts a destructive influence on the surrounding lung tissue, with subsequent hemoptysis, pulmonary abscess, or bronchiectasis.

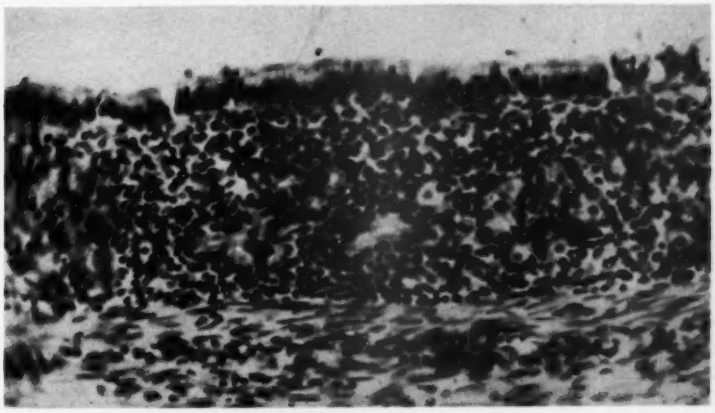

Fig. 6.—Gastric mucosa in the cyst wall. Cylindrical epithelium, glandular tubules, and a few parietal cells are seen.

On the other hand, there was some objection against performing a big thoracic operation on such a young child. It was thought preferable to postpone the operation until the child was a little older. It was then decided to discharge the child and to readmit it if and when symptoms made further postponement of operation impossible. On Sept. 10 the patient was discharged, and two days later he was readmitted because of an intercurrent infection; this cleared up in one week with penicillin therapy. During this week one puncture was done.

The third hospitalization was on Feb. 28, 1949, when the patient was 13 months old and weighed 8,980 gm. The general condition was fairly good, but symptoms due to the cyst were now much more pronounced, with frequent cough and difficult swallowing. Aspiration was again performed, and the fluid obtained again contained 16 units of a proteolytic ferment (Fuld's method). In March the patient was transferred to the surgical department and was operated on by Prof. Nuboer, who successfully removed an elastic tumor the size of a tennis ball. Adhesions were found between the cyst wall and the spine and the periosteum of the second to fourth ribs at the costotransversal articulations. Blood vessels came from the spine and from the mediastinum. The cyst was covered with pleura. By displacement of the lung an abscess was revealed in the apical segment of the lower lobe of the left lung. The upper lobe was atelectatic. After removal of the cyst the lung expanded satisfactorily. It then became evident that in the left lung, the lower lobe was well developed whereas the upper lobe was unusually small.

The tumor was examined by Dr. de Minjer (Institute of Pathology; Head, Prof. P. Nieuwenhuijse). The wall consisted of two layers of strong muscular tissue; in the inner lining cylindrical epithelium with glandular tubules and a few parietal cells were found (Fig. 6).

After the operation the patient developed normally, in keeping with his age. The roentgenogram of the thorax now showed no abnormalities, neither did the bronchogram of the left lung.

In addition to the intrathoracic cyst with gastric mucosa in the wall, this patient also had vertebral anomalies. The transverse processes of the fifth and sixth cervical vertebrae were fused on the right side. The neural arches of the seventh cervical and first thoracic vertebrae were not closed. These anomalies were visible on ordinary roentgenograms (Fig. 7) but were clearer on tomograms. Because of these findings and because we had been struck by the reported high frequency of vertebral anomalies occurring together with intrathoracic gastrogenic cysts, the entire spinal column was examined tomographically. This revealed the following: The vertebral bodies of the fifth, sixth, and seventh cervical and first and second thoracic segments had longitudinal defects and slight malformations.

In order to detect the presence of other skeletal abnormalities, the entire skeleton was examined roentgenologically, but no other congenital malformations were found.

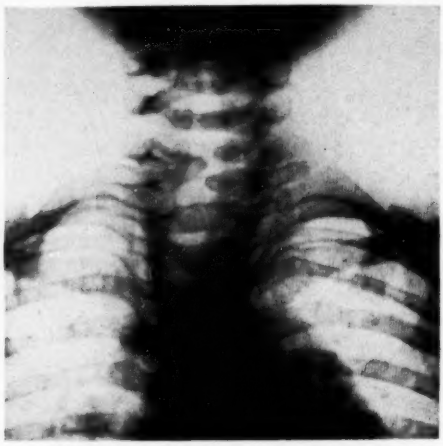

Fig. 7.—Vertebral anomalies. The transverse processes of the fifth and sixth cervical vertebrae are fused on the right side; the neural arches of the seventh cervical and the first thoracic vertebrae are not closed.

Because of the possibility of hereditary abnormalities, roentgenography was done on the spinal columns and the chests of both parents and two siblings, but no abnormalities were found. Identical findings in another patient are published elsewhere.

COMMENT

About 30 cases of this type of cyst have been described, the last 15 since 1940, of which 13 have appeared in the American literature.[1a]

The cysts are situated near the spine, and it is often reported that there are strong adhesions between cyst and spine.[1] Less often pressure atrophy of one or

1a. Davis, E. W., and Salkin, D.: J. A. M. A. **135**:218-221 (Sept. 27) 1947.

1. (*a*) Black, R. A., and Benjamin, E. L.: Enterogenous Abnormalities: Cysts and Diverticula, Am. J. Dis. Child. **51**:1126-1137 (May) 1936. (*b*) Brass, K.: Frankfurt. Ztschr. Path. **50**:26-33, 1936. (*c*) Olenik, J. L., and Tandatnick, J. W.: Congenital Mediastinal Cysts of Foregut Origin, Am. J. Dis. Child. **71**:466-476 (May) 1946. (*d*) Poncher, H. G., and Milles, G.: Cysts and Diverticula of Intestinal Origin, ibid. **45**:1064-1078 (May) 1933. (*e*) Roth, M.: Virchows Arch. path. Anat. **86**:371-390, 1881. (*f*) Steele, J. D., and Schmitz, J.: J. Thoracic Surg. **14**:403-406, 1945.

more ribs was noted. There was sometimes fixation to, but never communication with, the esophagus. Right-sided cysts are three times more numerous than left-sided ones. There was equal distribution between the sexes. The size varies in diameter from a few centimeters to the distance from the top of the lung to the diaphragm.

The thickness of the wall appears to vary between 1 and 5 mm. Serosa was never found. The muscularis consists of two, sometimes three, muscular layers, in which nerve cells and fibers are seen not infrequently. The submucosa may present inflammatory and hemorrhagic signs. The mucosa may be folded and have all the characteristics of gastric mucosa. Sometimes it is present only in a few scattered areas of the cystic wall.

The color of the cystic fluid varies among light yellow, ochre, brown, and green. The consistency is that of water or syrup; it is either opalescent or turgid. The reaction is neutral or acidic. Hydrochloric acid and pepsin are often, but not always, demonstrable. Four times a peptic ulcer was found with perforation.

Other congenital anomalies have been described in association with this congenital abnormality.[2] Most frequent are those of the vertebrae. Scoliosis is mentioned frequently, less often hemivertebrae and incomplete fusion of the neural arches, and there is a single case of spina bifida.

A few instances of abdominal enteric cyst or diverticula in combination with the above-mentioned intrathoracic cysts are described. In the literature the occurrence of intrathoracic cysts in association with vertebral anomalies is considered to be an "incidental" and unrelated finding.[3] Simultaneous occurrence of intrathoracic and abdominal cysts is thought to be related. In the majority of cases the presence of these cysts was revealed at or immediately after birth or at a very early age, only a few being discovered after the fourth year. The presence of secreting gastric mucosa is probably the factor which decides when the first symptoms will appear.[4] It follows, therefore, that the latter may be absent. If present, they can be ascribed to two causes. They are either due to pressure on adjoining organs, the lungs with the bronchi, esophagus, and great vessels, or they arise from peptic ulcer perforating into the surrounding tissues. In the first case the symptoms may be dyspnea, cyanosis, cough, atelectasis, inflammation, fever, and dysphagia; in the second case, inflammation, bronchiectasis, and hemoptysis.[5] In both cases the general condition may suffer. The preponderance of right-sided cysts is explained by the fact that the stomach during embryonic development also rotates toward the right,[4] or by assuming that the aorta would impede a left position.[3]

Various theories have been expounded to explain the occurrence of intrathoracic gastrogenic cysts. In general, it may be said that the strength of developmental potency in gastrointestinal mucosa is such that the histological structure of the cystic mucosa is of little value in determining its origin.[1d] The innervation is of greater

2. (*a*) Davis, E. W., and Salkin, D.: Intrathoracic Gastric Cysts, J. A. M. A. **135**:218-221 (Sept. 27) 1947. (*b*) Guillery, H.: Zentralbl. allg. Path. **69**:49-53, 1937. (*c*) Olken, H. G.: Am. J. Path. **20**:997-1009, 1944. (*d*) Schmincke, A.: Virchows Arch. path. Anat. (supp.) **227**:12-27, 1920. (*e*) Black and Benjamin.[1e]

3. Ehler, A. A., and Atwell, S.: J. Thoracic Surg. **17**:809-820, 1948.

4. Schwarz, H., II, and Williams, C. S.: J. Thoracic Surg. **12**:117-130, 1942.

5. Böss, C.: Virchows Arch. path. Anat. **300**:166-179, 1937. Ladd, W. E., and Scott, H. W., Jr.: Surgery **16**:815-835, 1944. Seydl, G. N.: Frankfurt. Ztschr. Path. **52**:346-[illegible], 1938.

importance. Lewis and Thyng [6] have been able to demonstrate diverticula in the alimentary tract of mammalian and human fetuses during embryonic development. Regression of these diverticula before birth is the rule, but persistence may lead to the development of enteric cysts.

Keith [7] gave the opinion that development of duplications of the alimentary tract is comparable to that of atresia, in that the development of one single lumen fails to occur, in a transverse direction where duplications are concerned, in a longitudinal direction in cases of atresia. This hypothesis was supported by Bremer.

Black and Benjamin [1a] consider these cysts to be intrathoracic remnants of the omphalomesenteric duct (vitelline duct). Poncher and Milles [1d] point out that the dorsal mesentery with its blood vessels is well developed at the time of the obliteration of the vitelline duct, from which these cysts are supposedly derived. The diverticula or the cysts should be found between the folds of the dorsal mesentery, vascularized by the mesenteric vessels. This may be true for abdominal cysts, but it is hardly possible in the case of intrathoracic ones. Olenik and Tandatnick [1c] express the opinion that "all the cysts are derived from the foregut at or near the region of the lung buds," which cannot be denied.

On quite a different level, and not at all in contradiction with the views mentioned above, is the hypothesis that the organizer substance must have influenced the development of this anomaly (Schwarz and Williams [4]). However, too little is known of this "organizer" to use it as a valuable argument.

Up to now, stress has been laid on the association with the foregut or the esophagus. Personally, I should like to center the interest on the spine. It is apparent from the literature that adhesions with the spine are more frequent than those with the esophagus. Of all congenital anomalies occurring together with these cysts, developmental anomalies of the vertebrae, especially the upper thoracic vertebrae, were seen most frequently.[2c] Moreover, in some of the roentgenograms reproduced, vertebral anomalies are visible which are not mentioned by the authors.[8] Finally, we learned that there are vertebral anomalies which may be found only on tomography and which remain undiscovered on the ordinary roentgenograms. Longitudinal defects in vertebral bodies and arches especially are more numerous than would be suspected from inspection of ordinary roentgenograms. It is hardly probable that a congenital anomaly of such frequency would be of no importance whatsoever in the development of another congenital anomaly: the intrathoracic gastrogenic cyst. Possibly the primary disturbance must be looked for not in a developmental anomaly of the foregut, but in an accident involving the spine and the foregut together at a time that their cells still adhere to each other. It might be supposed that for an unknown reason notochord and foregut fail to separate in the normal way, but remain in connection, and in this area cysts or diverticula of the stomach and foregut might develop. This appears to occur not very rarely. Patten [9] says "fusions of the notochord at its cephalic end . . . with pharyngeal epithelium are not uncommon occurrences."

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6. Lewis, F. T., and Thyng, F. W.: Am. J. Anat. **7**:505, 1907.

7. Keith, A.: Human Embryology and Morphology, Baltimore, William Wood & Company, 1933; cited by Davis and Salkin.[2a]

8. Davidson, L. R., and Brown, L.: J. Thoracic Surg. **16**:458-467, 1947. Poncher and Milles.[1d]

9. Patten, B. M.: Anat. Rec. **95**:307-311, 1946.

It is understandable that if the stomach moves caudally over a distance of from the fourth cervical segment, where its origin is, to below the diaphragm, a part of the foregut still fused with the notochord may fail to participate in this movement and lose contact with the stomach. But it also follows that in this part of the notochord there may arise anomalies presenting as anomalies of the vertebral bodies. This conception explains the fact that sometimes a connection with or below the diaphragm persists.[10]

Enteric cysts and diverticula, although less frequent, probably owe their development to the same mechanism. Since in this area a descent, as described for the stomach, does not occur, large deformities of the vertebrae are unlikely. This conception does not explain the defects in the neural arches, unless it were possible to demonstrate that the anomalies in the arches are secondary to those of the vertebral bodies or notochord.[1a]

SUMMARY

A patient with an intrathoracic gastrogenic cyst is described. The cyst was successfully removed, and the patient remained in good condition subsequently. Certain vertebral anomalies which were additionally noted were studied tomographically. This method revealed more longitudinal defects in the vertebral bodies and arches than would have become visible on ordinary roentgenological examination. These anomalies were chiefly situated in the lower cervical and upper thoracic regions of the spine.

The vertebral anomalies described in the literature showed the same predilection. The frequent occurrence of intrathoracic gastrogenic cyst together with certain anomalies of the cervical and thoracic parts of the spine makes it seem possible that a relationship exists between the two.

The theory is advanced that adherence of notochord and foregut persists. Descent of the stomach would then cause anomalies in the vertebral bodies (notochord) in this part of the spine, while the corresponding part of the stomach would be pinched off.

10. Dickson, J. A.; Clagett, O. T., and McDonald, J. R.: J. Thoracic Surg. **15**:318-323, 1946. Valle, A. R., and White, M. L., Jr.: Ann. Surg. **123**:377-383, 1946.

Correspondence

CHANGE IN ORAL EXAMINATIONS

To the Editor:—As examiner in pediatrics for the National Board of Medical Examiners, I should like to bring to the attention of the pediatricians an important change in Part III of the examination, as it involves pediatrics. This is the oral examination given at the end of a year's internship by "subsidiary boards" in twenty-nine medical centers throughout the country. The clinical nature of the examination provides an opportunity to determine the candidate's ability to apply the knowledge which he has previously demonstrated in the written examination of Parts I and II.

For many years the examination has included medicine, surgery, and obstetrics as major subjects, while pediatrics has been included in a group of specialties as being of minor importance. The examination has been revised, and pediatrics has been placed with the major subjects, where it belongs. In the revised form an oral bedside examination will be given during one full day. Two hours are assigned to medicine, two hours to surgery, one hour to pediatrics, and one hour to obstetrics.

It is expected that approximately 1,500 candidates will take the examination in June in the various centers. If each examiner examines six hours (six candidates) on the day of the examination, it will require 500 examiners in medicine and in surgery, and 250 in pediatrics and in obstetrics. While the men in the other three groups are accustomed to the examination, it is a new experience for pediatricians. The number of examiners needed at each center will depend on the number of candidates presenting themselves for the examination at the particular center. All details as to the time, place, and who will be asked to assist in the examination are in the hands of the local subsidiary board at the center.

It further means that the wards of children's hospitals and children's wards in general hospitals must be used to obtain material for the purpose of bedside examination. Previously the examination has not called for the use of patients in the pediatric examination.

As many pediatricians are aware, it has taken years of continuing effort to have pediatrics recognized as one of the four major branches of clinical medicine. It was not until the mid-thirties that pediatrics was given a separate examination by the National Board of Medical Examiners in Part II of their examination. Now that the place of pediatrics has been fully recognized, it becomes the responsibility of the pediatricians, and of the children's hospital services, in the various centers to cooperate fully and wholeheartedly with the local subsidiary boards in the centers where the examinations will be held.

Borden S. Veeder, Examiner in Pediatrics of the National Board of Medical Examiners, St. Louis.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

Alvah L. Newcomb, M.D., *President*

Regular Meeting, Feb. 20, 1951

Fetal Erythroblastosis in a General Hospital. Dr. Herbert F. Philipsborn Jr. (inaugural thesis), Glencoe.

This paper analyzes data obtained from 302 Rh-negative women who delivered consecutively 305 infants at the Evanston Hospital during the period from May 10, 1949, to Nov. 20, 1950. During this period there was a total of 3,470 deliveries. The fact that only 302 (8.4%) of the women were Rh-negative is not due to the greater incidence of spontaneous abortions in women with this blood type nor to erroneous typing.

Twelve infants of this group required treatment with blood for the syndrome of fetal erythroblastosis (hemolytic disease of the fetus and newborn). During the same period, two infants of Rh-positive mothers manifested this syndrome and required treatment with blood. Nine of the twelve treated infants born to Rh-negative mothers were boys, and three were girls. Five of the boys and two of the girls were born of secundipara secundigravida mothers.

The initial erythrocyte counts, hemoglobin values, and per cent of nucleated erythrocytes did not vary appreciably among nonerythroblastotic infants and infants showing slight to moderate serologic evidence of the disease without clinical manifestations. All tests were made shortly after birth. The sex of the infant and the parity or gravidity of the mother did not appear to affect the hematologic picture of those infants not clinically ill.

The birth of an erythroblastotic infant should be anticipated from the maternal history, and appropriate laboratory studies should be made during pregnancy. When such an infant is born, the cord blood should be studied immediately; the erythrocyte count, hemoglobin value, and Rh-type of the infant may be determined from this sample of blood. The criterion for the type of therapy has evolved as experience has increased. It is our feeling that we are treating sick infants, not sensitized blood. As a consequence, our criteria are (1) replacement blood transfusions for infants needing blood in the first 12 to 24 hours, (2) single or multiple small transfusions for infants needing blood in the neonatal period but after the first 24 hours of life, and (3) no treatment for those infants, regardless of whether the erythrocytes are sensitized, whose blood cell count does not fall or whose cell count drifts slowly to a level of 3,000,000 and then stabilizes. Blood cell counts are taken two or three times in the first 24 hours, then daily, and then at increasingly longer intervals until the red cell count is stable.

In this group there was one patient, inadequately treated, who died with kernicterus. Anemia did not develop in this infant in spite of a strongly positive reaction to a Coombs test and progressive jaundice. His case is evidence that a rigid set of rules for treatment may be a source of therapeutic failure.

Diagnosis of the Infantile Type of Coarctation of the Aorta: Report of Seven Cases. Dr. Benjamin M. Gasul, and Dr. Egbert H. Fell.

The paper is based on a study of seven cases of infantile type of coarctation. Slides illustrating the hemodynamics of the condition were shown, with roentgenographic and electrocardiographic findings. Clinical diagnosis may be made on the basis of these data.

Case 1.—A newborn infant in heart failure, with marked cardiac enlargement, was first seen in 1938. No clinical diagnosis was made at that time. When he was 2 years old, the diagnosis of adult type of coarctation of the aorta was made. A successful operation was performed in 1950, and the coarctation was removed. A review of the roentgenograms and electrocardiograms made in 1938 proves this to be a possible infantile type, followed by closure of the patent ductus arteriosus and subsequent development into the adult type of coarctation.

CASE 2.—A newborn infant with right heart failure was first seen in 1948. The clinical diagnosis was probable isolated myocarditis. Autopsy proved this to be a case of infantile type of coarctation of the aorta.

CASE 3.—A 9-day-old infant with right heart failure was seen in 1949. The clinical diagnosis of infantile type of coarctation was confirmed at autopsy.

CASE 4.—A 2-month-old infant with acute paroxysmal auricular tachycardia and heart failure was seen in 1949. The clinical diagnosis of infantile type of coarctation was confirmed at autopsy.

CASE 5.—A very cyanotic infant, 2 weeks old, was seen in November, 1950. The infant's fingernails were very cyanotic, and the toes were definitely noncyanotic. A clinical diagnosis of complete transposition of the aorta with coarctation of the infantile type was made. Autopsy confirmed the diagnosis.

CASE 6.—A 2-week-old cyanotic infant was seen in 1949. A diagnosis of tricuspid atresia, complete transposition of the great vessels, and a possible infantile type of coarctation of the aorta was made. Autopsy confirmed the diagnosis.

CASE 7.—In January, 1951, a 1-month-old infant was admitted to our service. Following complete examination and study, including an angiocardiogram, a diagnosis of coarctation of the aorta was made. The autopsy showed a combined adult and infantile type of coarctation.

There is a great deal of misunderstanding in the literature with regard to coarctation of the aorta. Abbott stated that coarctation in infants is of little clinical importance. Taussig has an excellent chapter on coarctation of the aorta in her monumental book on congenital malformations of the heart. Others have stated that the diagnosis is not important, because the infantile type is usually associated with other grave cardiac malformations and is, therefore, rapidly fatal, while the adult type is hardly a factor in the causation of death. In three of our cases the condition was not associated with other cardiac malformations, and we have recently studied a 3-month-old infant for whom we made a diagnosis of adult type of coarctation which was confirmed at autopsy.

Calodney and Carson of St. Louis published an important article in the July, 1950, issue of *Pediatrics*, covering a review of cases seen at St. Louis Children's Hospital and Washington University School of Medicine between 1938 and 1949. They described 14 cases of infantile type, 5 of combined type, and 2 of adult type, all in infants. The correct clinical diagnosis was made by them in a number of cases, and they stated that "a careful survey of the literature has failed to reveal a single case report of coarctation diagnosed in the newborn or young infant and corroborated by autopsy findings." We believe that this diagnosis has probably been made by other clinicians and that they have failed to report their cases, just as we have failed to report our cases until now. We believe also, as they do, that "with the recent advances in great blood vessel surgery, it may be possible to operate on and save a few of these infants who ordinarily have no chance to survive."

The essential difference between the infantile and the adult type of coarctation lies, we believe, in the fact that the hemodynamics of the infantile type corresponds closely to that of the fetal circulation. In the adult type, because of the fact that the coarctation is below or just at the point of entrance of the ductus arteriosus, the fetus must develop a collateral circulation in order to survive, and, after birth, the infant with the adult type is usually able to get along very well because of this collateral circulation. The differentiation of the two types is of importance only as long as the ductus remains patent. If the ductus closes it makes no difference where the coarctation is located, as far as the hemodynamics are concerned.

In the infantile type of coarctation of the aorta, the electrocardiogram usually shows right ventricular hypertrophy or combined hypertrophy.

Summary.—Seven cases of infantile type of coarctation of the aorta have been presented. Three were of noncyanotic type, three of cyanotic type, and one of combined infantile and adult type. The diagnosis can easily be made by regularly palpating the femoral arteries, recording blood pressures of the upper and lower extremities, and making roentgenologic and electrocardiographic studies. Although surgery has never been attempted in these cases, we believe that surgical intervention is indicated for some of these infants and may be lifesaving.

Breast Feeding in Private Practice. Dr. E. Robbins Kimball (inaugural thesis), Evanston.

The breast-feeding efforts of the mothers of 409 infants managed by me from birth to the age of 10 or more months are compared with those of 596 infants not managed consistently by me. The latter are used as a control group. In the management of nursing mothers several features were emphasized. 1. All mothers were expected to try to nurse; 3% refused. 2. The mothers were taught the technique of hand expression of colostrum and breast milk. 3. In order to prevent sore nipples the period of nursing was limited to one to three minutes, depending upon the shape of the nipple, the complexion, and the parity. 4. Both breasts were nursed at each feeding. 5. Complementary formula was used only in 1- or 2-oz. (30- to 60-gm.) quantities in the neonatal period and only if, in the opinion of the head nurse, the babies cried too much; 14% received some formula in the neonatal period. 6. A schedule for rest was outlined for every mother leaving the hospital. She was encouraged to drink beer, wine, or a cocktail before her rest periods, which numbered three for the first month, and two subsequently for as long as she nursed. 7. The nursing schedule was almost completely self-demand during the first few weeks at home. 8. Cereal feeding with a spoon was started as early as after two weeks if the baby seemed hungry. It was given irregularly and in varying amounts.

When leaving the hospital, 93% of the mothers were nursing; at one month 70% were still nursing; at two months, 53%; at three months, 46%, and at six months, 23%. This was 15 to 27% more breast feeding than in the control group at the same respective ages.

Sixteen mothers had breast infections associated with high fever. All received hot wet compresses promptly and were encouraged to nurse frequently from the infected breast. Eight received penicillin. In five cases the infants were weaned, and four mothers of these infants required surgical drainage for one to two weeks after weaning. In the 11 who continued to nurse none required surgical drainage. In the control group breast infection was cause for prompt weaning, and several mothers gave a history of having had surgical drainage.

Usually weaning was gradual; most mothers took about a month to wean their infants. Worry and fatigue of the mother, rather than illness in either mother or infant, caused most of the weaning.

DISCUSSION

Dr. Willard Z. Kerman, Glencoe: I am impressed by Dr. Kimball's results. He probably has four times as many mothers breast feeding as the general average for his community. It is important to remember that a mother's attitude toward breast feeding is determined not only by the prevailing opinions of her cultural milieu, by the comments of her friends, and by things she reads in magazines but also, and more particularly, by deep-seated emotional determinants, lifelong in their origin. For this reason, although mothers may be encouraged to breast feed, attempts to coerce them may be harmful as well as futile in some cases.

One of the chief benefits of breast feeding is usually overlooked; that is, the satisfaction and pleasure a mother obtains therefrom. Her emotional benefit may be equal to that of the child. There has been abundant psychoanalytic research which indicates that the establishment of breast feeding affords an opportunity for recreating the symbiotic relationship which exists during gestation and which accounts for the euphoria of the pregnant woman. The postpartum depression which sometimes follows has been ascribed in part to interruption of that relationship.

I am unable to accept the concept that physical fatigue is in itself responsible for loss of breast milk. In those cultural environments where women work the hardest there is the highest percentage of breast feeding. This being true, I fail to see how afternoon rests in bed can prevent loss of milk. I would like to reemphasize the point that breast feeding is advantageous not only to the baby but also to the mother and that pediatricians should stress this in encouraging mothers to breast feed their infants.

Dr. Eugene T. McEnery: I would like to compliment Dr. Kimball on this presentation of a subject that is always interesting. Several years ago Dr. R. Blessing read a paper before this society in which he analyzed the same situation. He found the mean age of the mothers he studied to be between 25 and 30; I wonder if Dr. Kimball's series were younger or older.

Dr. Gerard N. Krost: Did Dr. Kimball mean to imply that nursing the baby lessens the danger of surgical intervention when an infection already exists?

DR. S. C. HENN: I always approach this subject with a positive attitude, because we know it is nature's way. I think it a mistake to compare a mother to a cow, because I do not think they work the same way. I question whether nursing from both breasts is a good thing in all cases; when weaning is desired, it is best to nurse from both. I have always been in favor of mothers having beer or anything that increases body fluids, thus increasing breast milk.

There is considerable difference in the equipment mothers have. One with poor nipples makes a mistake in trying to nurse her baby. I think it a mistake to use an electric pump or other methods of the sort to induce milk, because the trauma is not good for the breast. Mothers with poor equipment are always most desirous of nursing their infants, and we have adhered to the policy of letting them try it until they are satisfied that it cannot be done. Sometimes we have difficulty in getting them to try because they have taken a negative attitude from the obstetricians who have told them that there is not much chance of their being able to nurse. We would probably have more success if obstetricians would encourage mothers to work on the breasts prior to delivery. In many cases the breasts might be made more capable of producing through massage with cacao butter.

I favor the idea of not-too-vigorous nursing at first. I have seen too many cases in which vigorous nursing resulted in no milk at all. I also commend hot dressings if there is infection of the breast.

DR. E. ROBBINS KIMBALL, Evanston: Dr. Kerman mentioned the danger of coercion in getting a mother to nurse. I do not know how to do that. All I do is explain, as persuasively as I can, the advantages of nursing and try to instill in the mother confidence that she can nurse. Even if she does not want to, or thinks she cannot, she will usually change her mind if she can be persuaded to persist for two months. Many women have been glad that I insisted upon their making the attempt. Further than that, I am not enough of a psychologist to know whether or not it does any harm. I have yet to see such a result. Charlotte Naish is in a good position to know about breast feeding, being the mother of five and a physician of experience, and she believes it does no harm to urge mothers to nurse. She believes that a woman who does not want to nurse did not want her baby in the first place; therefore, if allowed to wean her baby, a sense of guilt is added to her resentment. In several instances when I have persuaded mothers to nurse against their will, I believe it has made them talk more freely concerning their resentment, which, of course, is good for them.

In speaking of fatigue, many women have told me they had been advised to stop nursing because it was tiring. I think it is not the breast feeding that tires the mother, it is the other things that she thinks she has to do.

Dr. McEnery asked about the age of the mother in relation to her ability to nurse. We have not gone over our material carefully enough to give a scientific answer, but I do not believe we would find anything of statistical value as to age. I know women do not like to be compared to cattle, but it is well known that cows improve with successive calving up to a point, and then they seem to wear out. I have had good luck with older mothers, however, perhaps because they are more settled and will follow directions better. I have several multiparous and a few primiparous mothers in their forties who have nursed their infants successively. In practice, I believe that if they are not too old to have a baby they are not too old to nurse.

With regard to the amounts and types of alcoholic beverages prescribed, beer and wine are the favorites. I should say that one drink before each nap or two or three drinks a day would be about right. Two grandmothers, however, scolded me for prescribing liquor for their daughters-in-law.

Dr. Krost asked about breast infection. Again, it was Charlotte Naish who started me thinking about breast abscess. It is more frequent during upper respiratory infections, and it usually occurs from the fourth to eighth week post partum, whether the baby is off the breast, nursing with complementary feedings, or completely breast-fed. It occurs at the time when extra food is usually prescribed, when the mother's lactating capacity is still fluctuating, and it is difficult to give extra foods in such amounts that the infant drains all the lobules at each feeding. In this state of imbalance some of the lobules become plugged, and the breast becomes tender, painful, and finally inflamed. Because of pain the mother frequently omits nursing from the tender breast, which augments the imbalance further, whereas frequent nursing and hand expression

would open the plugged ducts and reverse the process. I intended to imply that prompt action, including frequent nursing and administration of antibiotics, would tend to prevent the ultimate necessity for surgical drainage.

With reference to Dr. Henn's remarks concerning one-breast versus two-breast feedings, I wish to say that we almost always recommend the use of both breasts at each feeding. Here again, we recall the farmer who would not think of milking only one side of a cow's udder. Since most of the breast milk is obtained in the first two to four minutes of sucking, and since the most frequent problem is insufficient quantity, it behooves us to see that the infant gets as much as he can at each feeding. Hence, I should agree with Richardson, who states that the proponents of one-breast feedings usually have a small percentage of patients nursing. The one-breast idea brings up again the thought of emptying the breast. One cannot empty the breast, because one can get some milk indefinitely by hand expression. It would seem better to think in terms of reducing the intralobular pressure, which permits more secretion of milk.

Concerning the small and inverted nipples, I should like to point out that Wandsborough in England developed a metal nipple-shield which looked like a small tin hat. It was worn over the nipple and held in place by a brassiere. It exerted gradual and gentle suction on the nipple, which was usually sufficient, over many months, to cure the retraction. Recently in America a similar shield that is better looking has been made from plastic and is known by its patent name plastishield.® These are obtained from Plastishield, Inc., Minneapolis, and we now have them in our hospital. Whenever I find small or flat or inverted nipples I prescribe these for the mother to wear when not nursing or hand expressing. It seems to draw out the nipple so that the baby has a better chance to grasp it. The ideal procedure would be to have the obstetrician prescribe the plastishield® during pregnancy. In the two instances in which I witnessed this, the mothers were able to nurse their second children after having been unsuccessful with their first.

How much milk should a mother produce in order to consider nursing worth while? I think it takes two weeks for most mothers to learn how to nurse and six to eight weeks to become proficient. If a mother has really tried to become proficient and can produce 1 oz. per feeding, I believe it is worth her while. Again, I would like to emphasize that mental rest is very important.

Alvah L. Newcomb, M.D., *President*

Regular Meeting April 17, 1951

Residents' Essay Program

Use of Desoxycorticosterone Acetate in Dehydration and Malnutrition. Dr. Howard S. Traisman.

This paper was published in full, with Dr. John A. Bigler as co-author, in the November, 1951, issue of the A. M. A. American Journal of Diseases of Children, page 548.

Treatment of Infantile Diarrhea with a New Combination of Antibiotics. Dr. Elmer R. Kadison.

A study is presented of a series of 53 infants, under 1 year old, who were treated for infantile diarrhea with a combination of two antibiotics, neomycin and bacitracin, as compared with a control group who received sulfadiazine, streptomycin, and penicillin procaine, alone or in combination. The combination of neomycin and bacitracin used was "neobacin," and each tablet contained 10,000 units of neomycin and 2,000 units of bacitracin.

The first group consisted of nine patients who had amebiasis. Seven patients received one tablet every six hours for 14 days, and three of these received two tablets every six hours for 14 days. Two patients received 2,500 units of each drug every six hours for five days. The diarrhea subsided in all patients within one to four days. All were asymptomatic, but two still harbored Endamoeba histolytica in the stools.

The second group consisted of four subgroups. The first was composed of nine infants with diarrhea who were treated with "neobacin" alone. They were asymptomatic within an average of 3.22 days. The second subgroup, containing 13 patients who were treated unsuccessfully with other drugs and when given "neobacin," responded within an average of 2.88 days. Patients in

both the above series received one tablet of "neobacin" every six hours for four to seven days. The third subgroup consisted of 13 infants treated with "neobacin," in a dosage of one tablet every six hours for four to nine days, and penicillin procaine, 150,000 units intramuscularly, daily. They failed to respond to the other drugs, and all had nasopharyngitis or bronchopneumonia. The diarrhea subsided in all cases within four days. The last series consisted of nine infants who were treated with sulfadiazine, streptomycin orally, and penicillin procaine intramuscularly. The average duration of diarrhea was 6.44 days.

No toxicity resulted from the drug; the tablets were given with apple sauce or jelly and were well tolerated. Stool counts of the patients receiving "neobacin" showed marked depression of the intestinal flora.

Acute Infectious Gingivostomatitis (Herpetic Stomatitis) and Its Response to Oral Administration of Aureomycin. C. R. CASTALDI, D.D.S.

Acute infectious (herpetic) gingivostomatitis has been recognized as a disease entity for many years, but not until recent times has the herpes simplex virus been found to be the causative organism. Numerous and various methods of treatment have been prescribed. These include oxygen-liberating mouth washes, potassium chlorate, vitamin therapy, topical applications of silver nitrate, chromium trioxide, methylrosaniline chloride, and, within recent years, administration of the sulfonamide drugs and penicillin. Careful examination of the results of these methods revealed, however, that the infection appeared to run a similar course regardless of the treatment.

A study was done at Children's Memorial Hospital to determine whether aureomycin, an antibiotic of known antivirus activity, would prove to be of value in treating the infection. Aureomycin was administered in systemic dosages of 25 mg. per kilogram of body weight per day, divided into four equal doses. (The dispersoid form mixed with cold milk was found to be the best method of administration.) No additional treatment, such as acetylsalicylic acid or mouth washes, was used. Controls received placebos.

To date 42 patients have been treated with aureomycin, and 14 have been used as controls. In the treated group acute symptoms lasted on the average 2.07 days, with a median of 1.37 days, while in the untreated group acute symptoms lasted on the average 3.17 days, with a median of 2.5 days.

With regard to total duration of infection, the treated patients showed an average of 7.97 days, with a median of 7.5 days, while in untreated patients the condition lasted, on the average, 11.17 days, with a median of 9.5 days. Two family studies showed significant differences in untreated and treated patients.

It was noted that the range for acute symptoms and total duration was approximately the same in the two groups, showing that some of the treated patients did not respond to aureomycin.

Clinical Study of Amebiasis in Infants Under One Year Old. DR. CHARLES GOLDENBERG.

Although amebiasis in adults is now recognized as a not-uncommon disease in the United States, the infection in infants, particularly those under 1 year old, is thought to be relatively rare. However, after a one-year survey (July, 1949, to July, 1950) at Cook County Hospital, 80 infants with amebiasis were found among 872 cases of diarrhea in children, an incidence of 9.2%.

The importance of repeated parasitologic examinations of the freshly passed stool specimens is emphasized as the basis for the diagnosis of amebiasis. In addition, proctoscopy for more than 62.5% of the patients was found to substantiate the diagnosis with typical bowel patterns, which were briefly described. Proctoscopy aided in determining the progress of treatment and helped in deciding the ultimate prognosis.

Analysis of the 80 patients with amebiasis enabled us to classify our cases into four main groups, based upon the patients' clinical history and course of illness. The wide divergence of symptoms between each group so classified again illustrates the protean manifestation of amebiasis in infants. It is therefore stressed that amebiasis should be included in the differential diagnosis of gastrointestinal dysfunction in all infants under 1 year old.

A description of the specific antiamebic therapy of the 80 infants follows. The patients were divided into three comparable groups of 25, 30, and 25 infants and were treated with bacitracin, iodinated bacitracin, and diiodohydroxyquinoline (diodoquin®), respectively. Plain bacitracin and iodinated (13% iodine) bacitracin were used in tablet form, each tablet containing 10,000 units of the antibiotic. Infants weighing less than 7,000 gm. were treated with 10,000 units of bacitracin, or iodinated bacitracin per day for 14 days, while those weighing more than 7,000 gm. were treated with 20,000 units per day for 14 days. The dose of diiodohydroxyquinoline was 2 grains per kilogram of body weight per day for 14 days. The three drugs were compared as to efficacy in producing clinical improvement, in eliminating Endamoeba histolytica from the stools after cessation of therapy, and in their respective ability to produce a permanent cure during a three-month follow-up period.

Our study showed that in 49 infants, or 61.5% of all patients, stools contained no amebae at the completion of therapy. In only 36 infants, or 45%, the stools remained free of amebae during the three-month follow-up period. The need for more specific antiamebic drugs, as well as control of reinfections from other members of the family, was emphasized.

Respiratory Obstruction in Early Infancy: Report of Four Cases Due to Suprahyoid Thyroglossal Cyst. Dr. Donald Johns.

An important emergency in pediatrics is respiratory obstruction in early infancy. A discussion of the general problem of respiratory obstruction serves as a background for the reporting of four cases in which an unusual cause, a suprahyoid thyroglossal cyst at the base of the tongue, was encountered.

Contributing factors in obstruction to the airway in infants are the small dimensions of the infant larynx, the comparatively narrow laryngeal aperture, the peculiarity of the submucosal structures which easily provoke obstructive edema in inflammation or trauma, and the rigid cartilaginous ring of the cricoid which permits tissue to expand only at the expense of the airway.

This report discusses the various causes which may be classified as congenital anomalies, laryngeal paralysis, neoplastic disease, and inflammatory processes. The causes lack similarity, but they have the common symptomatology of respiratory embarrassment.

The embryology and pathology of thyroglossal cysts are discussed in detail. These lesions, which are retention cysts in a patent portion of the vestigial thyroglossal tract, may occur anywhere from the base of the tongue to the region between the hyoid bone and the thyroid gland. They are far commoner below the hyoid than above it; rarely does the cyst form at the base of the tongue.

Four cases were reported, in which classic examples of the syndrome of respiratory obstruction were presented in three infants, ages 6 weeks, 6 weeks, and 10 days, respectively. The infants showed dyspnea, occasional cyanosis, retractions, stridor, and dysphagia. The other patient, aged 2½ years, had only mild dysphagia. The diagnosis in all was made by lateral roentgenograms of the neck, which showed in each a mass at the base of the tongue which projected into the pharynx posteriorly; these findings were confirmed by direct laryngoscopy.

All the patients were treated conservatively. The cysts were aspirated through an infant laryngoscope, without anesthesia. The procedure was followed by immediate subsidence of symptoms. Gradual recurrence of symptoms with reaccumulation of fluid in the cyst was seen in each case. Repeated aspiration resulted in gradual disappearance of the lesion. This form of therapy was followed because of the poor prognosis these patients presented.

Embryonal Adenomyosarcoma: A Twenty-Year Survey. Dr. H. Jack McDonald.

The purpose of this survey was to review all cases of embryonal adenomyosarcoma (Wilms's tumor) at the Children's Memorial Hospital over a 20-year period from Jan. 1, 1930, to Jan. 1, 1950, and to evaluate diagnostic measures and treatment. A brief review of similar surveys made at other medical centers was presented.

Twenty-nine patients were included in the survey. An analysis of each case included data regarding the patient's age, sex, date of admission, chief complaints, duration of symptoms, pre-

operative and postoperative irradiation, date and side of nephrectomy, review of pathologic reports and slides of surgical specimens, metastases, intravenous pyelogram, urinary findings, course, and other facts pertinent in each specific case.

Follow-up investigations were made in all cases, and 8 of the 29 patients were found to be alive and doing well, a survival rate of 27.5%. Survival period for the eight living patients varied greatly. All had been living for at least 2 years with no evidence of metastasis at the time the survey was made, and one patient had been living 16 years since initial diagnosis and surgery. Over this 20-year period, during which there was some disagreement as to the preferred method of management, it was natural to find a variety of forms of therapy. All patients treated at Children's Memorial Hospital underwent nephrectomy unless metastases were found. In addition, all patients received immediate postoperative irradiation. Sixteen of the twenty-nine patients also had preoperative irradiation. It is interesting to note that only two of the eight living patients had received preoperative irradiation. The preferred current treatment consists of nephrectomy with immediate postoperative irradiation.

The relative merits of transperitoneal incision over other surgical approaches for nephrectomy were discussed briefly. The differential diagnoses were also mentioned.

The importance of early diagnosis cannot be emphasized too much. The prognosis seemed to be related to the age of the patient at the time diagnosis was made. It was an interesting fact that in 5 of the 29 cases studied the condition was discovered upon routine physical examination.

Abstracts from Current Literature

Biochemistry; Bacteriology and Pathology

EXPERIMENTS WITH FILTRATES OF B. COLI. K. BLECKMANN, Ztschr. Kinderh. **66**:195, 1949.

Filtrates of several strains of Bacterium (Escherichia) coli were injected intracutaneously in a group of children with and without gastrointestinal symptoms. The test resembled in its principle the tuberculin reaction. The results were interpreted macroscopically as well as with the capillary microscope. The author hopes by this method to have found a new way to investigate E. coli infections and the reaction of the individual. GLASER, Jerusalem, Israel.

Metabolism; Infant Feeding; Milk and Other Foods

CONTENT OF FREE AMINO-ACIDS IN THE CHILD'S ORGANISM. J. STRODER, H. GRUNHOFER, and W. ENGEL, Ann. pædiat. **177**:304 (Nov.) 1951.

Using a slight modification of the Pope-Stevens method, the authors tested the concentration of free amino acids in the blood of healthy and sick children and babies. The following findings are reported: 1. In the course of infectious fevers there were no changes from the normal of between 5 and 8 mg. per 100 cc. 2. After encephalography there was usually a drop in the concentration which later rose to normal again. A similar effect was observed after administration of epinephrine and insulin. 3. The content of amino acid in the blood of persons suffering from gastric indigestion corresponds to normal, while in toxicoses an initial increase is observed. 4. The kidneys are able to concentrate and dilute amino acids. The degree of amino-aciduria may be dependent on the water excretion in cases in which the amino acid concentration remains constant in spite of increased water excretion. The maximum concentration of urinary amino acids occurs four hours after oral administration of protein. When protein was given with and without the addition of carbohydrates, it was found that more amino acids were excreted in the latter case.

FROM THE AUTHORS' SUMMARY.

HUMAN MILK LIPASE. E. FREUDENBERG, Bull. schweiz. Akad. med. Wissensch. **7**:396 (Oct.) 1951.

The lipase of mothers' milk in its natural medium is a completely inactive enzyme in relation to its natural substrate, the milkfat. It is not activated in the manner described by Willstatter for other lipases.

In 1927 the author found bile acids, the derivatives of dioxycholane and trioxylcholane, and scymnol, as well as neutralized gastric juice, to be activators.

The effect of gastric juice and of the extracts of the mucous membrane is not based on a kinase but on the lipase of the stomach. Certain intermediary products, diglycerides and monoglycerides may, probably, be directly disintegrated by the lipase of mothers' milk.

FROM THE AUTHOR'S SUMMARY.

IODINE AND FLUORINE AND THYROIDS IN SWISS SCHOOL CHILDREN. V. DEMOLE, Bull. schweiz. Akad. med. Wissensch. **7**:430 (Oct.) 1951.

The daily administration of a fluorinized complement of 0.25 to 0.3 mg. of fluorine (fluoride sodium tablets) to 194 school children in Berne and to 182 school children in Moudon, Switzerland, for a period of 18 to 22 months, has not produced any noticeable modification of the thyroid gland.

The thyroid glands of the school children of Chippis (Valais), an industrial zone of hyperfluorosis, and of those from Sembrancher (Valais), where the drinking water contains 1.47 mg. of fluorine per liter, are of normal dimensions. The population of these localities, situated in a region which had been goitrous, has overcome the thyroid problem by the consumption of iodized salt (5.0 mg. of iodide of potassium per kilogram); there is also an anomalous high fluorine occurrence.

These facts show that the theory of the antagonism between iodine and fluorine is of no value. It is advisable to practice prophylaxis against dental caries by means of a fluorine complement.

FROM THE AUTHOR'S SUMMARY.

CHRONIC DISTURBANCES OF CALCIUM AND PHOSPHATE METABOLISM IN CHILDREN. G. FANCONI, Schweiz. med. Wchnschr. **81**:908 (Sept. 22) 1951.

Different forms of chronic hypocalcemia are discussed, such as pseudohypoparathyroidism and hypoparathyroid cretinism. Hypocalcemia in the course of phosphatemias is described, with special emphasis on a new entity, the "renal phosphatediabetes" with hyperphosphaturia and hypophosphatemia, which can occur alone or combined with a chronic aminoaciduria with or without cystinosis, and which occasionally occurs also with renal glycosuria. A new syndrome with chronic hypercalcinemia is characterized by chronic hypercalcemia and hyperphosphatemia and low alkali reserve, osteosclerosis, craniostenosis (early closure of the cranial sutures), oligophrenia, persisting nitrogen retention, and low phosphate clearance.

AMBERG, Rochester, Minn.

TOTAL AMINO ACID DETERMINATION IN BLOOD AFTER ORAL ADMINISTRATION OF PROTEIN HYDROLYSATE (NESMIDA NESTLE). ILSE ANTENER, Schweiz. med. Wchnschr. **81**:970 (Oct. 6) 1951.

A colorimetric method for determination of total amino acid in blood is described, dependent upon the color reaction of the sodium salt of beta-naphthoquinone sulfonic acid with amino acids in alkaline solution (Folin method). The standard solutions for comparison were prepared with a mixture of aminoacetic acid and proline. Certain substances, such as sulfonamides, give the same reaction. This may have to be considered in case of medication. The fact that satisfactory results are obtainable with 0.1 ml. of blood is of advantage in infants and children.

AMBERG, Rochester, Minn.

INFLUENCE OF NEGATIVELY-IONIZED AIR ON THE LACTEAL SECRETION IN HYPOGALACTIC NURSING MOTHERS. CARLO CAMMARELLA, Riv. clin. pediat. **49**:613 (Sept.) 1951.

Fourteen nursers with hypogalactasis have been treated with negative aeroionization (inhalation of negatively ionized air).

Secretion of milk increased more than 30% in seven cases; an increase not significant from a practical standpoint was observed in four cases; no beneficial effect was observed in the remaining three cases.

Further effects have been observed on hunger, nervous system, psychical functions and blood formation. It can therefore be stated that negative aeroions surely have a biological action on the organism, a stimulating and invigorating action on vital activity.

FROM THE AUTHOR'S SUMMARY.

THE HYPOGLYCEMIC SYMPTOM COMPLEX IN INFANCY AND CHILDHOOD. FRIEDRICH MEYTHALER and ROSEMARIE FISCHER, Deutsche med. Wchnschr. **75**:69 (Jan. 19) 1950.

Hypoglycemia in infants and children is discussed in general.

AMBERG, Rochester, Minn.

TREATMENT OF DISTURBANCES OF NUTRITION IN INFANTS AND CHILDREN. BANNURAH, Med. Klin. **38**:1013 (Sept. 21) 1951.

Infants and young children with severe enteritis and with dyspepsia were treated with streptomycin and dihydrostreptomycin, with good results.

AMBERG, Rochester, Minn.

CONTRIBUTIONS TO THE KNOWLEDGE OF IRON AND COPPER METABOLISM IN CHILDHOOD: PART III. W. BRENNER, Ztschr. Kinderh. **66**:299, 1949.

Further studies on iron and copper metabolism were made on a group of diseases with low serum iron and normal serum copper levels, in which iron therapy seemed indicated. In this

group were anemia of hemorrhage, nutritional hypochromic anemia, celiac syndrome, and certain forms of anemia in leukemia, nephrosis, and congenital heart disease. A second group comprised diseases with elevated iron and normal copper levels, including hemochromatosis, pernicious anemia, hemolytic anemia, Banti's syndrome, and malnutrition. The third group consisted of diseases with elevated levels of both metals and included acute myeloblastic leukemia and panmyelophthisis.

GLASER, Jerusalem, Israel.

INFLUENCE OF ORAL AND INTRAVENOUS HUMAN SERUM INTAKE ON NITROGEN BALANCE. H. PLÜCKTHUN, Ztschr. Kinderh. **66**:496, 1949.

Metabolic balance studies with human serum given orally or injected intravenously were performed on infants. Results showed that with intravenous infusion, in cases in which carbohydrate and fat intake was adequate to cover caloric needs, nitrogen retention varied from 90% in infants with extreme protein deficiency to 50% in infants not suffering from protein starvation. Such good utilization could not be obtained by oral administration of human serum, although here, too, the retention of nitrogen was higher than with human milk or cow's milk as the source of protein. This high utilization is explained by the high biological value of human serum protein in comparison with other proteins.

GLASER, Jerusalem, Israel.

THE USE OF NITRITE DETERMINATIONS BY MEANS OF THE GRIESS REAGENT TO DETERMINE THE PRESENCE OF BACTERIUM COLI IN MOTHER'S MILK. I. PFEILSTÜCKER, Ztschr. Kinderh. **66**:596, 1949.

Bacterium coli changes sodium nitrate, added for test purposes to mother's milk at milk-collecting stations, into sodium nitrite. Sodium nitrite with Griess' reagent gives a red color, which can be used for quantitative and qualitative determinations of the Bact. coli content of milk.

GLASER, Jerusalem, Israel.

LATENT DAMAGE DUE TO PROTEIN DEFICIENCY IN CHILDREN. H. EWERBECK, Ztschr. Kinderh. **67**:85, 1949.

A study of school children, 6 to 12 years of age, who had been found healthy on physical examination, showed that, although the total protein content of the plasma had not diminished, the globulin fraction, especially the gamma globulin fraction (antibody carriers), was greatly reduced. The author believes that a simultaneous diminution of the water content of the blood is the cause for the qualitatively normal protein level. He points out that neither physical examination nor simple protein determination (without fractionation) will reveal latent malnutrition which may cause permanent damage to the children. The cause of malnutrition is not necessarily an insufficient total protein intake but may be an insufficient animal protein intake in the presence of relatively high total protein intake.

GLASER, Jerusalem, Israel.

Vitamins; Avitaminoses

CORRELATION BETWEEN VITAMINS AND VITAMIN DISEQUILIBRIUM. V. BISCEGLIE, Bull. schweiz. Akad. med. Wissensch. (Supp. 1) **6**:5 (March) 1950.

The existence of reciprocal relationships among the different vitamins has now been confirmed by experimental results and clinical observations. Not only is the presence of the different vitamins in the body fluids and tissues indispensable, but it is equally essential that they be present in well-adjusted proportions. Failing this, the lack of one vitamin may have repercussions on the others and thus give rise to serious imbalances. Thus, it may be proved that an excess of one factor provokes the rapid exhaustion on another vitamin, leading to a "deficiency without deficiency." This phenomenon would be due not to the absence of a certain vitamin from the diet but to the diminution or disappearance of a factor through its being more rapidly eliminated or in some way rendered unusable as a result of the excess of another vitamin. Thus, the repeated administration of strong doses of thiamine can cause symptoms of pellagra, and repeated high doses of nicotinic acid can lead to the appearance of symptoms of polyneuritis and

ariboflavinosis. Several other observations regarding interrelationship between the components of the B group have been made (between thiamine and pyridoxine, between folic acid and thiamine, etc.). Relationships have been shown to exist not only between the members of the B group but also between vitamin C and several factors of the B complex, between hydrosoluble and liposoluble vitamins, and finally, between liposoluble vitamins.

From observations gathered in this way, it follows that a balance exists between the various vitamins and that the absence of or increase in one factor is not without effect upon the consumption and requirement of other vitamins.

On the other hand, since vitamins take part in enzymatic systems, it can be shown that an elevated dose or a lack of a certain vitamin participating only in a single enzymatic system disturbs the coordination of the other systems.

The recognition of the reciprocal relationship between the vitamins and of vitamin imbalances (altered ratios between the various vitamins) is of both scientific and practical importance, since it touches upon the domains of therapeutics and of dietetics.

FROM THE AUTHOR'S SUMMARY.

BIOLOGICAL ANTAGONISMS AND DEFICIENCY SYNDROMES DUE TO DISEQUILIBRIUM OF VITAMINS. V. CHINI, Bull. schweiz. Akad. d. med. Wissensch. (Supp. 1) **6**:20 (March) 1950.

Clinical observation shows the existence of deficiency states appearing in subjects treated with other vitamin factors, usually in excessive doses. Thus, symptoms of nicotinic acid deficiency can be observed in subjects treated with high doses of thiamine and vice versa. One may speak of a true "vitamin antagonism due to disequilibrium" and hence of a deficiency syndrome due to a vitamin disequilibrium.

These antagonisms are found particularly among the vitamins of the B complex, which usually act synergistically. They also exist between the B vitamins and vitamin C, and thus between vitamins which take part in the cellular oxidation-reduction processes. This may aid in explaining the phenomenon, although the explanation still remains obscure.

The problem has been studied experimentally in a systematic manner for the first time by Maluzzi-Valeri and his co-workers (Perosa, Conese, Putignano, Neri, Ramunni). These authors have thus established a solid experimental basis for the study of these disequilibrium antagonisms.

Numerous clinical facts observed during vitamin treatment can be explained by this "doctrine." These observations provide a certain foundation, in the light of which one may also try to interpret the results recently reported during folic acid therapy (aggravation or appearance of symptoms of funicular myelosis, polyneuritis, and pellagra). The study of these unfavorable effects, considered from this angle, enables one to understand better the mechanism of the action of folic acid and liver extracts, and will perhaps provide a more exact interpretation of the so far obscure pathogenesis of combined degeneration of the posterior cords of the medulla.

These ideas have a practical application; they should serve as a guide in vitamin therapy, during which, especially with vitamins of the B complex, every attempt must be made to avoid excessive disequilibrium, which may have unfavorable consequences.

This fact also follows from the experimental observations of Malaguzzi-Valeri and Perosa, according to which a deficiency syndrome (e. g., pigeon beri-beri), favored by a vitamin disequilibrium (excess of vitamin B_1, pellagra-preventing factor, etc.) is very resistant to, and often unaffected by, treatment with the specific vitamin (B_1). This fact is difficult to explain.

FROM THE AUTHOR'S SUMMARY.

TREATMENT OF RICKETS WITH PARENTERAL VITAMIN D_2. HANS ROOS, Monatsschr. Kinderh. **98**:457 (Nov.) 1950.

Complete cure of rickets was obtained by one intramuscular injection of 1.25 mg. of vitamin D_2, corresponding to an oral dose of 30 mg. Onset of healing was slower with the intramuscular route than with the oral. This makes the intramuscular method undesirable in the treatment of rickets complicated by spasmophilia.

HARTENSTEIN, Tokyo, Japan.

PREVENTION OF RICKETS IN PREMATURE INFANTS BY ONE INTRAMUSCULAR INJECTION OF SMALL DOSES OF VITAMIN D. MARIE LUISE KENN, Monatsschr. Kinderh. **98**:479 (Nov.) 1950.

Forty premature infants each received one intramuscular injection of 6,250 units of vitamin D_2 or D_3 during the first week of life and were followed clinically and by x-ray examination for six to eight months. Rachitic epiphyseal x-ray changes occurred in one infant who had recieved vitamin D_2 and in seven infants who had received vitamin D_3. Prophylactic intramuscular administration of one dose of vitamin D_3 to premature infants is advocated as a result of these studies. This method, however, is contraindicated in the treatment of rickets because of the slow absorption of intramuscularly administered vitamin D.

HARTENSTEIN, Tokyo, Japan.

DETERMINATION OF ALKALINE PHOSPHATASE IN SERUM AND ITS SIGNIFICANCE IN THE DIAGNOSIS OF RICKETS. O. HÖVELS, Ztschr. Kinderh. **66**:237, 1949.

The author describes in detail a laboratory method requiring smaller amounts of serum for the determination of alkaline phosphatase. His normal values are slightly higher than those of Bodansky, which are usually considered as standard.

GLASER, Jerusalem, Israel.

Hygiene; Growth and Nutrition; Public Health

INFLUENCE OF MODERN THERAPEUTICS ON INFANT MORTALITY. D. SEROPEO PAJARES GARCIA, Acta pediat. españ. **9**:803 (July) 1950.

Garcia discusses the lag between knowledge and performance in lowering the infant mortality. His discussion is from the viewpoint of a health officer. There is a wide difference between the death rate in cities and in rural areas. In the rural centers, even where there are clinics, the clinics are not manned by physicians trained in the care of children. Better courses in pediatrics and preventive medicine are obligatory if this situation is to be ameliorated. Older physicians should be encouraged by stipends to take time off for study of the use of newer drugs and methods of treating children. Better nutrition, widespread immunization against preventable diseases, and use of sulfonamides and antibiotics would lower appreciably the death rate in infancy, but progress is being made along these lines at a very slow rate. A campaign for the education of the lay people is also very essential if infant mortality is to be lowered.

ALLEN, San Francisco.

THE CHANGES IN BIRTH WEIGHT IN THE LAST FIFTY YEARS: COMPARISON OF GERMAN CLINICS WITH GYNECOLOGICAL HOSPITAL IN BASEL. K. SOLTH and K. ABT, Schweiz. med. Wchnschr. **81**:58 (Jan. 20) 1951.

During the last 50 years 204,333 birth weights were at the disposal of the authors. Of these, 115,868 were obtained from three German clinics (Berlin, Marburg, Würzburg) and 88,456 from Basel. In all there was an increase in birth weight in the course of the last 50 years as well as a decrease during war years. With the age of the mother up to about 35 years the birthweight increased. This increase was less pronounced in the Basel material than in that of the three German clinics. This was the only difference. The birth weights of primiparae and multiparae and the sex in relation to birth weight did not differ much. It is of interest to note that the unknown influences on the birth weights were operative in Basel as well as in the German material, although Basel was not directly involved in the war.

AMBERG, Rochester, Minn.

THE INFANT MORTALITY RATE FROM INTESTINAL DISEASE IN RELATION TO THE AREAS SPRAYED WITH DDT AND OCTA-KLOR. S. CORBO, Arch. ital. di pediat. e puericolt. **13**:261 1949.

The child mortality rate caused by intestinal disease was investigated in the province of Latima, which was divided into two areas only one of which was sprayed with chlorophenothane

(DDT). In the treated area the mortality rate was lower whenever chlorophenothane had killed the flies.

The great importance of chlorophenothane also in the prevention of children's diseases has thus been proved. The author concludes by advocating its rational use together with chlordan ("octa-klor").

FROM THE AUTHOR'S SUMMARY.

GROWTH OF SCHOOL CHILDREN AFTER THE FIRST AND SECOND WORLD WARS (COMPARATIVE BODY MEASUREMENTS AND WEIGHTS OF SCHOOL CHILDREN IN MANNHEIM, GERMANY). W. STEPHAN, Kinderärztl. Praxis **17**:242 (July-Aug.) 1949.

Body measurements and weights of children in three age groups (6, 9, and 13 years) throughout the years 1912 to 1948 demonstrate graphically the influence of nutritional disturbances following the world wars and the results of some of the relief measures.

GLASER, Jerusalem, Israel.

THE INFANTS' INSTITUTE OF OSTRAVA. H. MOHWALDOVA, Pediat. listy **5**:297, 1950.

A discussion is given of the scope of the institute, its care for infants up to 2 years of age and the mothers who need medicosocial attention, and its function as a center of disease prevention and as an institute for the education of health workers, medical personnel, and doctors. A review of its activities during the years 1930-1950 is presented, with accompanying diagrams.

FROM THE AUTHOR'S SUMMARY.

Prematurity and Congenital Deformities

THE WEIGHT PROGRESS OF PREMATURE INFANTS GIVEN SUPPLEMENTARY FEEDINGS OF VITAMIN B_{12}. HENRY RASCOFF, ALLA DUNEWITZ, and ROBERT NORTON, J. Pediatrics **39**:61 (July) 1951.

The addition of vitamin B_{12} to the diet of premature infants does not increase their weight gain.

MACDONALD, Pittsburgh.

CONGENITAL DEFICIENCY OF ABDOMINAL MUSCULATURE WITH ASSOCIATED GENITOURINARY ABNORMALITIES: A SYNDROME; REPORT OF NINE CASES. J. FREDERICK EAGLE, JR., and GEORGE S. BARRETT, Pediatrics **6**:721 (Nov.) 1950.

Nine cases of congenital deficiency of the abdominal muscles with obstructive manifestations of the genitourinary tract are presented. The "syndrome occurs" practically always in a male and the testes are almost invariably in the abdomen. Other frequently associated anomalies are patent urachus, pigeon-breast deformity, and talipes equinovarus.

The muscle deficiency varies from mere weakness to complete absence and is easily recognizable at birth. When such a defect is noted, extensive investigation of the genitourinary tract is mandatory, because in all reported cases there is at least an enlarged bladder and there may be bilateral hydronephrosis and hydroureter. Urethral obstruction has been present in most of the recently reported cases, and it is suggested that "neurogenic imbalance" may account for the remaining cases. It is felt that the muscular defects are secondary to urinary obstruction in utero.

Treatment should be directed toward early surgical correction of the urinary tract obstruction and stasis.

FROM THE AUTHORS' SUMMARY.

STUDIES ON PREMATURITY: IV. INFLUENCE OF FETAL MATURITY ON WEIGHT LAG PERIOD. MORRIS STEINER and WILLIAM POMERANCE, Pediatrics **8**:513 (Oct.) 1951.

During the course of an investigation of factors influencing mortality of premature infants, it was noticed that the initial lag in weight gain of infants varied greatly and appeared to be unrelated to the birth weight of the infant. The present study was undertaken to determine the relationship of maturity and birth weight to initial loss and gain of weight of the premature infant (weight lag period). The weight lag periods of 623 premature infants with birth weights

ranging between 1,000 and 2,250 gm. were studied. It was found that the time required for a premature infant to regain its birth weight and continue to gain was inversely related to the length of its gestation period.

The importance of considering gestation period (maturity) in studies of premature infants is emphasized.

FROM THE AUTHORS' SUMMARY.

OSTEOCHONDRODYSTROPHIA DEFORMANS (MORQUIO BRAILSFORD DISEASE). N. FELDMAN and M. E. DAVENPORT, Arch. Dis. Childhood **26**:279 (Aug.) 1951.

Four cases of eccentro-osteochondrodysplasia (osteochondrodystrophia deformans, Morquio's disease) are described. Two of the cases are in persons of native African stock and two are in persons of mixed European-African racial origin. These seem to be the first reported cases of this disease in persons not of purely European racial origin. Consanguinity of the parents is present in three cases. The blood chemistry investigations show no significant departure from normal. A close relationship exists between the many different varieties of dyschondroplasia.

KELLY, Milwaukee.

ACTION OF VITAMIN B_{12} ON INCREASE OF IMMATURITY. MICHELE PARADISO and EUCLIDE MONDANI, Riv. clin. pediat. **49**:590 (Aug.) 1951.

The authors tested the action of Vitamin B_{12} in premature infants and noted its therapeutic efficacy, thus confirming the results of experimental researches made by others on animals. The authors consider the possible mechanism of action of the vitamin.

FROM THE AUTHORS' SUMMARY.

TWO CASES OF OCULAR HYPERTELORISM (GRIEG'S DISEASE). C. ELGRDI CALLJA and ARACIL MUNOZ, Acta pediat. españ. **8**:1069-1075 (Sept) 1950.

Two cases of hypertelorism are presented, in children 18 and 17 months respectively. Both were mentally and physically retarded and showed x-ray evidence of Grieg's disease. One child was mildly rachitic, and the sphenoid bone was deformed, with intracaruncular distances of 34 cm. and 40 mm., respectively. The sinuses of the sphenoid were greatly changed in shape, but the sella turcica was small. The eye fundi were normal in both cases. These last findings are most important because they differentiate these cases from Crouzon's disease.

Malformations of fingers, toes, and other long bones are sometimes noted in Grieg's disease as well as anomalies of soft tissue, such as cryptorchidism.

The disease is sporadic, although some cases appear to be familial. Syphilis, consanguinity, and aged parents have been blamed, but there is no conclusive evidence that any of these are etiological factors.

ALLEN, San Francisco.

GARGOYLISM AND HURLER'S SYNDROME. MIGUEL FIGUEROA·M., Rev. chilena pediat. **21**:31 (Jan.) 1950.

Two cases of lipochondrodystrophy, or gargoylism, are reported. These are the first two cases to be reported in this country. Both patients had the typical symptomatology: macrocephaly, corneal opacity, dorsolumbar kyphosis, hepatomegaly, disproportionate dwarfism, mental retardation, and the typical roentgenological changes. These changes were more characteristic in the second case.

FROM THE AUTHOR'S SUMMARY.

CLEIDOCRANIAL-PELVIC DYSOSTOSIS OR PIERRE-MARIE-SAINTON-DISEASE. LORENZO EXPOSITO, G. ROJAS, and G. CABRALES, Rev. cubana pediat. **20**:679 (Nov.) 1948.

A case of cleidocranial-pelvic dysostosis is reported in a baby of 21 months. There were delayed closing of the cranial sutures, bilateral absence of the clavicular diaphysis, and much retardation in the development of pelvic ossification centers. Other bones of the hip were underdeveloped for the child's age, and the secondary center of ossification of the femoral epiphysis was very small.

This combination of abnormalities is very rare; only two similar observations were found in the literature.

KEITH, Rochester, Minn.

PRENATAL NUTRITION AND CONGENITAL ANOMALIES. HERMANN SCHROEDER, Deutsche med. Wchnschr. **75**:351 (March 17) 1950.

It is concluded that proper nutrition of the mother during the first months of pregnancy is necessary for the infant.

AMBERG, Rochester, Minn.

LUSCHKA'S MALFORMATION OF THE RIBS. H. SCHÄFER, Kinderärztl. Praxis **17**:144 (March-April) 1949.

An asymmetry of the thorax in a 7-year-old boy led to the study of roentgenograms of the ribs, which revealed a fork-like split of the anterior part of the fourth rib. Differential diagnostic considerations of other malformations and of pathologic disturbances of the ribs leading to asymmetries in the thorax wall are discussed.

GLASER, Jerusalem, Israel.

PATHOGENESIS OF ANEMIAS OF PREMATURELY BORN INFANTS. F. KÜSTER, Ztschr. Kinderh. **65**:591, 1948.

The author divides "anemia of the premature" into two phases; the first phase is a very early anemia occurring up to the fourth week and is due to immaturity of the blood-forming bone marrow and the sudden arrest of extramedullary blood formation. This is aggravated by a shorter life expectancy of erythroid cells produced extramedullarily. There is no marked disturbance of the erythroid-myeloid ratio at this stage. The larger number of young basophilic erythroblasts points toward the increased activity of the blood-forming organs rather than toward a maturation anomaly. Beginning with the sixth week of extrauterine life (second phase), erythropoiesis increases, and with a lowering hemoglobin content, maturation variations become noticeable, such as increased numbers of mitoses with some pathologic forms. It seems, however, that these disturbances of maturation occur earlier (second to third month) than the time that the reservoirs of iron should be exhausted, which the author explains by a retention of iron by liver and spleen with an only gradual and too slow release. There is evidence for the beneficial effect of iron therapy in this stage of anemia. In the first stage, however, no therapy seems indicated. The second phase may further be aggravated by the very rapid growth of the infant and by the occurrence of intercurrent infections. The author suggests, therefore, the early treatment (beginning with the sixth week of life) of premature infants with iron medication.

GLASER, Chicago.

BRACHYDACTYLIA AND CONGENITAL SMALL LENS. K. SEELEMANN, Ztschr. Kinderh. **67**:1, 1949.

A case of brachydactylia with congenital small ball-like lens (Marchesan's syndrome) is described. The author considers it a hereditary mesodermal malformation similar to arachnodactylia (Marfan's syndrome) but with opposite reaction of the tissues.

GLASER, Jerusalem, Israel.

TASKS OF PEDIATRICIANS IN THE TREATMENT OF HARELIP AND CLEFT PALATE AND IN SOLVING PROBLEMS RELATED TO THESE DEFECTS. F. BURIAN, Pediat. listy. **5**:263 (No. 6) 1951.

The tasks of pediatricians in the treatment of congenital harelip and cleft palate are outlined. In these defects, besides a failure of the parts (upper jaw, lip, palate) to unite, there is also a deficiency of tissue and a weakening of their growing capacity. As a result, the defects persist to adult life. The pediatrician must participate in working out the plan of treatment and its execution and follow the patient's development continuously. The necessity for cooperation between various specialists is emphasized, and also the necessity for establishing day nurseries and nursery schools for these children.

The tasks involved in other problems connected with these defects, such as the cause, the part played by heredity, the influence of environment, are then discussed. Organization of coordinated research in institutes and departments concerned with treatment, notification of patients, and organization of continuous care for patients are suggested. The results of research by P. Fogh-Andersen in Denmark, where there is national registration and control of the treatment of patients, are described. Wide research on such a basis would permit the elaboration of eugenic measures and may become the solution to the question of prevention.

FROM THE AUTHOR'S SUMMARY.

CONTRIBUTION TO THE MANAGEMENT OF PREMATURE INFANTS OF THE LOWEST BIRTH WEIGHT GROUP. KAREL ZNAMENACEK, Pediat. listy **5**:275, 1950.

Two surviving premature babies, with birth weights of 900 and 1,000 gm. respectively, are reported on after a follow-up period of 6 and 10 months respectively. The methods of management of premature infants as used by foreign authors are discussed and compared with the methods used at the premature unit of the University Infants' Hospital Prague-Podoli.

FROM THE AUTHOR'S SUMMARY.

SIMPLIFIED ROOM INCUBATOR FOR TREATMENT OF PREMATURE INFANTS. J. SVEJCAR, Pediat. listy **5**:289, 1950.

Management of premature babies weighing 1,500 gm. or more may very well be carried out in heated cubicles instead of in individual incubators. The heating may be installed in the form of automatically regulated electric heaters and controlled by means of a thermograph and a hydrograph. In this way it is possible to obtain an absolutely stabilized temperature and a fairly stabilized humidity and to cut down expenses on materials and staff.

FROM THE AUTHOR'S SUMMARY.

PREMATURE UNIT OF THE UNIVERSITY INFANTS' HOSPITAL PRAGUE-PODOLI. K. KUBAT, Pediat. listy **5**:293, 1950.

A description of the premature unit of the University Infants' Hospital Prague-Podoli is given: it's equipment as to space, furniture, instruments, material and staff, and the organization of its services in it. There are 20 beds (4 incubators) for infants and 10 beds for breast-feeding mothers in the unit.

FROM THE AUTHOR'S SUMMARY.

CARE OF PREMATURE INFANTS: TASKS AND POSSIBILITIES. K. POLACEK and G. SYROVATKA, Pediat. listy **5**:294 (No. 6) 1951.

An analysis of the main causes of mortality of premature infants on the basis of statistical material is given. Close relationship to birth and the importance of birth trauma are emphasized. The point of view is advanced that there is little hope of lowering the death rate, which amounts at least to one-fifth of all premature infants, in well-equipped institutes under the guidance of pediatricians. State-wide intensification of the basic care of premature infants may save about 1,500 to 2,000 children yearly.

Instructions are given regarding the care of premature infants in the country and regarding the establishment of district hospital centers for premature infants.

FROM THE AUTHORS' SUMMARY.

Newborn

ASPHYXIAL ATTACKS IN THE NEWBORN INFANT DUE TO CONGENITAL OCCLUSION OF THE POSTERIOR NARES: REPORT OF FIVE CASES. HARRY MEDOVY and I. H. BECKMAN, Pediatrics **8**:678 (Nov.) 1951.

Among the less common causes of acute respiratory distress in newborn infants is congenital occlusion of the posterior nares or choanae. This malformation may be unilateral or bilateral, and the obstruction may be membranous or bony or a combination of the two. Because it is

rare, it is frequently overlooked. The diagnosis can be simply made provided the possibility of the occurrence of the condition is kept in mind. Inability to pass a No. 8 F. catheter through the nares into the nasopharynx, with confirmation by instillation of iodized oil into the nares and by positional roentgenograms, indicates the presence of a choanal obstruction. Unilateral conditions may be treated surgically in later childhood. Bilateral malformation should be operated upon as soon as possible and artificial openings created. Five cases are reported, three of them bilateral.

FROM THE AUTHORS' SUMMARY.

FUNCTIONAL INTESTINAL OBSTRUCTION IN THE NEWBORN. ISABELLA FORSHALL, P. P. RICKHAM, and D. B. MOSSMAN, Arch. Dis. Childhood **26**:294 (Aug.) 1951.

Functional intestinal obstruction of newborn infants, affecting several members of one family, is described. The possible relationship of this condition to congenital dilatation of the colon (Hirschsprung's disease) and the treatment are discussed. It is postulated that functional intestinal obstruction in newborn infants, with or without a familial basis, is a definite clinical and pathological entity.

KELLY, Milwaukee.

ERB'S PALSY WITH PHRENIC NERVE INVOLVEMENT. S. HASENOHR, Clin. Rep. Adelaide Child. Hosp. **2**:54 (May) 1951.

A case of right-sided Erb's paralysis with paralysis of the right side of the diaphragm is discussed. The importance of roentgenograms in establishing this diagnosis in babies born cyanotic and dyspneic after a difficult delivery is stressed.

MCGUIGAN, Evanston, Ill.

SCARLET FEVER IN A NEWBORN. R. SACREZ, MLLE. HOERNER, P. BEYER, and M. LAGOUTIERE, Pédiatrie **38**:211 (No. 3) 1949.

A child born to a mother who had tonsillitis at the time of delivery and an exanthema the following day showed an exanthema on the third day of life and died on the fourth day. Blood cultures of the child yielded Streptococcus pyogenes, and postmortem examination showed infection of the umbilical vein and signs of a virulent septicemia with hemorrhagic exudates in the serous cavities.

GLASER, Jerusalem, Israel.

ISO-IMMUNIZATION FROM AN UNIDENTIFIED ANTIGEN. S. FOLENA and ANNA STEFANINI, Minerva pediat. **2**:556 (Nov.) 1950.

The second of two children of healthy parents had icterus gravis. The first child, 9 years old, had had a normal birth with no icterus. The mother's blood type was O Rh-positive; the father's and the child's were A Rh-positive. No incompatibility between the mother's and the child's blood could be found in the Rh-Hr, M-N, or P group systems. The mother had developed a titer in the serum of anti-A agglutinins of 1/640. This high titer suggests collateral sensibility to some unknown antigen. The child recovered completely from the icterus following several large transfusions (A and O blood donors) and an exsanguination transfusion from a female Rh-negative donor.

HIGGINS, Boston.

DEMONSTRATION OF FREE RH ANTIBODIES IN THE BLOOD OF NEWBORN INFANTS AND CONSEQUENCES FOR OBSTETRIC PRACTICE. HELMUT BERNDT, Deutsche med. Wchnschr. **75**:318 (March 10) 1950.

The mother was Rh-negative, the father Rh-positive. The second to fifth children died with icterus gravis or hydrops universalis. The sixth child also had icterus gravis but responded to transfusion. During the seventh pregnancy no Rh antibodies could be demonstrated up to the eighth month. At the end of the ninth month agglutinating and blocking antibodies of low titer were found, with a slight increase at the beginning of the tenth month. A full-term girl became definitely icteric 10 minutes after birth, but responded to several transfusions. After birth the blood of the mother had a blocking titer of 1 : 1,024, the agglutinating antibodies were low as before. From the cord, tied immediately after birth two samples of blood were taken.

The first was examined immediately, the other after being kept four hours, first at room temperature and then in an incubator. The serum of the first sample had a blocking antibody titer of 1:512. In the other no antibodies could be demonstrated.

AMBERG, Rochester, Minn.

PENICILLIN PROPHYLAXIS FOR BLENNORRHOEA NEONATORUM. E. DARUP, Med Klin. **44**:1156 (Nov. 2) 1951.

Two drops of an aqueous solution of penicillin containing 2,500 units, per milliliter was instilled twice into the eyes of each of 2,667 newborn infants. No gonorrheic infection occurred, and nonspecific irritations were noted in 0.9%. Penicillin eye drops are to be regarded as the best method of Credé prophylaxis.

AMBERG, Rochester, Minn.

Acute Contagious Diseases

ISOLATION OF POLIOMYELITIS VIRUS FROM THE HEART IN FATAL CASES. C. W. JUNGEBLUT and J. E. EDWARDS, Am. J. Clin. Path. **21**:601 (July) 1951.

Jungeblut and Edwards, in investigating the etiology of the myocarditis of poliomyelitis, attempted virus passage studies on fragments of medulla, spinal cord, and heart in the five fatal cases and of the liver in one case. One milliliter of a 10% suspension of tissue was injected intracerebrally into pairs of young Cynomolgus monkeys. Poliomyelitis virus was isolated from the spinal cord and heart in two cases, from the cord only in the third case, from the heart only in the fourth case, and not at all in the fifth case. Two strains of virus from the cord were pased through monkeys several times. Only one strain from the human heart could be passed. A majority of the paralyzed monkeys showed myocardial lesions.

On the basis of this evidence, the authors submit the hypothesis that the myocarditis seen in poliomyelitis is due to the virus itself.

FOLEY, Boston [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

THE RISK OF POLIOMYELITIS AFTER TONSILLECTOMY. GAYLORD W. ANDERSON, GENEVIEVE ANDERSON, AUDRY E. SKAAR, and FRANZISCA SANDLER, Ann. Otol. Rhin. & Laryng. **59**:602 (Sept.) 1950.

A series of 21 cases of poliomyelitis in Minnesota occurring within one month after tonsillectomy during the summer of 1946 is reported. In all, 2,881 cases of poliomyelitis were officially reported in this epidemic. Of these, 2,709 supplied detailed epidemiological data. In the entire series, 226 patients died.

A report is made of 19 of the 21 cases occurring within one month after tonsillectomy. There were three deaths. In 12 patients (63.2%) bulbar poliomyelitis developed, as contrasted with 4 (22.2%) of 18 who had tonsils removed in the second or third month preceding onset of the disease. There was a concentration of cases of bulbar poliomyelitis the onset of which after the operation was within the usual range of incubation periods.

The authors concluded from this study that the risk of poliomyelitis was at least three times as great among patients undergoing tonsillectomy as among a comparable group not undergoing this operation, and the risk of bulbar infection was 11 times as great.

STEELE, Kansas City, Kan. [A. M. A. ARCH. OTOLARYNG.].

VIROLOGY OF POLIOMYELITIS. CHARLES F. PAIT, California Med. **73**:391 (Nov.) 1950.

The virus of human poliomyelitis has been demonstrated in excretions before the onset of the disease, during the disease, and during convalescence. It may be confused with different viruses likely to be found in the same sources in clinical conditions resembling poliomyelitis.

Children are likelier to excrete the virus than are adults, and the stools of young family contacts of patients are likelier to contain the virus than are the stools of adults of the family. Immunologic differences between strains of poliomyelitis virus have been detected, so that three types are now evident. The distribution of these types and their importance as causes of epi-

demics are not known. This multiplicity of immunologic types is an important factor in considering immunization of humans. Commercial manufacture of vaccines faces many technical problems.

Recently the Coxsackie virus has been demonstrated in humans with a disease closely resembling poliomyelitis.

PRYOR, Redwood City, Calif.

FORTY-TWO CASES OF POLIOMYELITIS IN CENTRALIA, 1949. HERBERT J. LEVINE, Illinois M. J. **96**:178 (Sept.) 1949.

Forty-two cases of poliomyelitis were diagnosed and confirmed in private practice in a five-week period in Centralia, Ill., from July 3, 1949, to Aug. 6, 1949. The age of the series of patients ranged from 3½ months to 28 years.

BARBOUR, Peoria, Ill.

EMBRYONIC HEARING ORGANS AFTER MATERNAL RUBELLA. LEROY A. SCHALL, M. H. LURIE, and G. KELEMEN, Laryngoscope **61**:99 (Feb.) 1951.

The incidence of deafness in the progeny after the mother has had rubella during pregnancy is not definitely established. However, almost all such progeny have some kind of congenital defect if the rubella was contracted early in pregnancy.

There have been few pathological investigations. The authors present reports of two cases with pathological studies of the temporal bones. Hemorrhage and hyperemia were conspicuous. Other authors, too, have reported hemorrhage. It is difficult to tell how much is due to trauma and how much to rubella. The embryonic development of the organs was normal. This is to be contrasted with Carruthers' case and the organs of the waltzing guinea pig.

Rubella may cause deafness by two methods: (1) an arrest in the development of the organ of Corti and (2) vascular changes. The latter may possibly be causative of the former.

HITSCHLER, Philadelphia [A. M. A. ARCH. OTOLARYNG.].

MECHANISM OF EMERGENCE OF RESISTANCE TO STREPTOMYCIN OF H. PERTUSSIS AND H. PARAPERTUSSIS DURING TREATMENT WITH THIS ANTIBIOTIC. HATTIE E. ALEXANDER and WINIFRED REDMAN, Pediatrics **4**:461 (Oct.) 1949.

In large populations of Hemophilus pertussis and H. parapertussis which have never been exposed to streptomycin, it is possible to demonstrate the presence of cells resistant to 100 γ of streptomycin per cubic centimeter.

These highly resistant cells exhibit the following traits which are characteristic of mutants: (1) irregular occurrence in different independent cultures of same population size and strain and therefore independence of the action of streptomycin; (2) transmission of the resistant trait unchanged in degree through many subcultures in the absence of streptomycin, and therefore inheritance of the trait; (3) a rate of occurrence consistent with mutation frequency; the mutation rate of cells resistant to 1,000 γ per cubic centimeter does not differ significantly between these two species.

FROM THE AUTHORS' SUMMARY.

HOW SUBSTANCES OBTAINED FROM "INSECTIVORE" PLANTS ACT ON SPASMODIC COUGH AND PERTUSSIS. T. GORDONOFF, Schweiz. med. Wchnschr. **81**:111 (Feb. 3) 1951.

Extracts prepared from Drosera rotundifolia L and of Pinguicula vulgaris L were tested with isolated rabbit intestine and guinea pig uterus which had been pretreated with acetylcholine, histamine, or barium chloride, the latter to test the effect on myogenous spasm. Extracts from Drosera effected a stronger neurogenous spasmolysis, those from Pinguicula a stronger myogenous spasmolysis.

AMBERG, Rochester, Minn.

STREPTOMYCIN AEROSOL TREATMENT IN PERTUSSIS. A. PISANI, Pediatria **58**:700 (Sept.) 1950.

One hundred fifty children (72 under 1 year of age) were treated daily with 200 mg. of streptomycin in an aerosol in three divided doses until the symptoms were relieved. When complications were present, penicillin and vaccine were also given. Of the 150 children, 134 showed complete recovery, 13 experienced some improvement, and 3 died.

HIGGINS, Boston.

NEED FOR A CAMPAIGN AGAINST PERTUSSIS IN BRAZIL. PEDRO RUEDA, Arq. Pediat. **23**:7 (Nov.) 1950.

The necessity of vaccination against pertussis in the United States is pointed out, and the need for similar action in Brazil is stressed.

CHRISTOPHERSON, Evanston, Ill.

ACTION OF URINE COLLECTED FROM CHILDREN RECOVERING FROM MEASLES. A. SEFRIN, Arch. Kinderh. **135**:8, 1948.

Fifteen to 20 cc. of urine collected from children on the 15th to the 18th day after the appearance of typical measles was given daily by enema, to be retained for at least one hour, from the 7th to the 11th day after certain exposure to measles. No protection or mitigation of measles was recorded.

ARON, Chicago.

TOXOID THERAPY OF DIPHTHERIA. J. STROEDER and T. HOCKERTS, Arch. Kinderh. **135**:71, 1948.

The toxoid therapy recommended by Cremer was tested in 33 children with toxic diphtheria (gravis type). The mortality was 48.4%, as compared with 41.1% in the control group of 51 children with toxic diphtheria not treated with toxoid. Toxoid therapy appears to be of no value.

ARON, Chicago.

AUGMENTED INCIDENCE OF POSTVACCINAL ENCEPHALITIS IN THE PROVINCE OF NORDRHEIN-WESTFALEN DURING 1946. WALTER GOETERS and ALFRED STECHERN, Arch. Kinderh. **135**:73, 1948.

During the year 1946, after smallpox vaccination, 21 children, 10 boys and 11 girls, all but one in large cities, became ill with meningomyeloencephalitic symptoms. All children were over 1 year of age; 17 of 21 were over 4 years of age; 19 children were vaccinated for the first time; 2 were revaccinated. Nine children died; eight were completely cured; three were nearly well, and recovery of one child is still uncertain. The duration of the disease varied from a few days to several months. The spinal fluid showed increase in protein and cells, predominantly lymphocytes, and there were elevated sugar values. Autopsies revealed hyperemia of the brain and dilatation of the veins and perivascular infiltration consisting of lymphocytes, histiocytes, and leucocytes. In one case demyelinization encephalitis with disintegration of the sheaths of the medulla was found.

ARON, Chicago.

TOXOID THERAPY OF POSTDIPHTHERIC PARESES. MARIANNE DRESCHERS, Arch. Kinderh. **137**:18, 1949.

One single intraspinal injection of 0.1 cc. of toxoid was given to two children, 4 and 5 years old; a third child 5½ years old received 0.25 cc. of toxoid intraspinally. The three children had severe and progressive pareses after diphtheria treated with antitoxin. As early as from the fourth day after the intraspinal injection the paretic muscles improved rapidly; the reflexes returned, and complete recovery was achieved ultimately. The dose of 0.1 cc. was tolerated without side-effects, while the dose of 0.25 cc. was followed by severe meningeal irritation and therefore is regarded as too high a dose.

The author is very enthusiastic about the instantaneous improvement that followed the intraspinal injection of toxoid in every one of the three cases.

ARON, Chicago.

RADICULAR MANIFESTATIONS IN POLIOMYELITIS. PAUL-HEINZ NEUMANN, Deutsche med. Wchnschr. **76**:107 (Jan. 20) 1951.

Some patients with poliomyelitis had persistent pains in the segments corresponding to the paralyzed parts with severe exacerbations at night and considerable increase of the proteins in the cerebrospinal fluid. Altogether, such findings are reported in 19 cases. Three of these concerned children 7, 9, and 13 years of age. Inflammation of the posterior roots is held responsible.

AMBERG, Rochester, Minn.

THE DIAGNOSTIC VALUE OF THE BLOOD-PICTURE IN PERTUSSIS. F. K. FRIEDERISZICK, Ztschr. Kinderh. **66**:229, 1949.

The author found in 623 cases of pertussis an increase above normal of white blood cells, with preponderance of lymphocytes in only 41.6%. The lymphocyte count is highest during the fourth week of the illness and disappears by the eighth week. The blood picture is more reliable as a diagnostic aid in children above 4 years of age, because of the absence of the physiologic lymphocytosis at this age. The observation of the blood count is more valuable as a means of observing the progress of the disease than as a diagnostic test.

GLASER, Jerusalem, Israel.

PENICILLIN TREATMENT OF SCARLET FEVER. JUSTUS STRÖM, Svenska läk.-tidning. **45**:369 (Feb. 27) 1948.

Every second patient with scarlet fever who was admitted to the Hospital for Infectious Diseases in Stockholm in 1946-1947 was treated with penicillin eight times a day for six consecutive days or more until the child was free from streptococci in the pharynx. There were 225 patients treated and 222 untreated controls. The commonest of the demonstrated 19 different kinds of streptococci were types 1 and 4. The length of fever was somewhat shorter, the bacterial complications markedly reduced, and there was less incidence of return in the treated group. An evident drawback was decreased immunity; there were five relapses after one to four and one-half months among the penicillin-treated group and none in the control group.

WALLGREN, Stockholm, Sweden.

STATISTICAL INVESTIGATION OF RESULTS OF DIPHTHERIA VACCINATIONS. D. HOOGENDOORN, Nederl. tijdschr. geneesk. **93**:3153 (Sept. 10) 1949.

As most children are vaccinated against diphtheria between the ages of 5 and 14 years, the numbers of diphtheria cases and deaths in this age group were compared with those of all other age groups in cities which could be compared, so far as the frequency among the different age groups was concerned. The cities were divided according to their vaccination condition into bad, fair, and good. The ratio between morbidity in the 5 to 14 age group and that in all other groups was for those three groups: 2.6, 1.6, and 1. The ratio between deaths in the 5 to 14 age group and deaths in all other groups was for the same groups: 4.5, 2.9, and 1. Those are statistically significant differences. If it had been possible to compare 100% vaccinated groups with entirely unvaccinated groups, the differences would have been even more striking.

VAN CREVELD, Amsterdam, Netherlands.

PENICILLIN THERAPY IN SCARLET FEVER. J. E. MINKENHOF, H. PEETERS and M. C. VISSER, Nederl. tijdschr. geneesk. **93**:3877 (Nov. 12) 1949.

The influence of penicillin in scarlet fever was studied. Four hundred and seventy-two patients were treated with 100,000 to 200,000 units per day for six days. This resulted in a reduction of the period of hospitalization to three weeks. Three times as many complications were observed in the control group as in the group of penicillin-treated patients. Two of the patients who were treated with penicillin had second attacks of scarlet fever after intervals of three and five months, respectively. There were two recurrences. In 131 cases throat swabs were taken regularly. In the majority of the cases the cultures for hemolytic streptococci became negative under penicillin therapy. Some cases, however, remained contagious. The treatment of scarlet fever with penicillin is recommended.

VAN CREVELD, Amsterdam, Netherlands.

Acute Infectious Diseases

VACCINIAL INFECTION IN CHILDREN WITH ATOPIC DERMATITIS. JOSEPH H. FRIES and STANFORD BORNE, J. Allergy **20**:222 (May) 1949.

The diagnosis of Kaposi's varicelliform disease was made for 21 children during the mass vaccination campaign in New York City in 1947. Immunologic studies were done on 16 of the 21 patients.

By neutralization and vaccination experiments, the vaccinia virus was proved to be the etiological agent. The atopic eczematoid skin is uniquely susceptible to virus invasion, particularly by the vaccinia virus. The varicelliform eruption of Kaposi, when due to vaccinia virus, and generalized vaccinia are different clinical entities.

HOYER, Cincinnati.

CLINICAL OBSERVATIONS ON TRICHOCEPHALUS TRICHIURUS (WHIPWORM) INFESTATION IN CHILDREN. RODNEY C. JUNG and PAUL C. BEAVER, Pediatrics **8**:548 (Oct.) 1951.

Of over 1,900 hospitalized children whose stools were examined routinely, 17.6% were infected with Trichuris trichiura (Trichocephalus trichiurus). The heavy infections (over 30,000 eggs per cubic centimeter), which consistently produced diarrhea and dysentery, were commonest in children 2 to 4 years of age. A significant correlation existed between worm burden and incidence of amebiasis.

A successful technic for the treatment of trichuriasis is described. The importance to the clinician of measuring worm burden is emphasized.

FROM THE AUTHORS' SUMMARY.

EFFECT OF ADRENOCORTICOTROPHIC HORMONE ON RHEUMATIC FEVER. ALBERT DORFMAN, KATHERINE SMULL, DELBERT M. BERGENSTAL, ROBERT HOWARD, RICHARD MULLER, DUNLAP OLESON, and EDWARD SENZ, Pediatrics **8**:603 (Nov.) 1951.

Eleven patients with chronically active rheumatic fever and eleven patients with acute rheumatic fever were treated with corticotropin (ACTH) for four to six weeks. Corticotropin therapy was found to repress the manifestations of active rheumatic fever, including those of carditis. There is as yet no conclusive evidence as to whether corticotropin therapy affects the natural course of rheumatic fever. A relatively large percentage of patients in this study showed evidence of reactivation of disease upon withdrawal of the drug.

As used in these studies corticotropin therapy did not uniformly prevent death due to acute rheumatic pancarditis. There is no evidence to indicate whether treatment of active rheumatic fever with corticotropin changes the incidence of late valvular deformities.

FROM THE AUTHORS' SUMMARY.

SERIAL ELECTROCARDIOGRAMS IN RHEUMATIC FEVER. B. GANS, Arch. Dis. Childhood **26**:315 (Aug.) 1951.

This paper emphasizes the importance and simplicity of making serial electrocardiograms for evaluating the progress of carditis in acute rheumatic fever. Although no individual change in the electrocardiographic curve is necessarily diagnostic, the occurrence of changes from record to record during the course of the disease is important in deciding how the patient should be managed. When there are no further changes in successive tracings, carditis has ceased. It is suggested that standard limb lead electrocardiograms be made daily during the early course of the disease and three times a week thereafter.

KELLY, Milwaukee.

SCARLET FEVER AND TONSILLITIS IN RELATION TO ACUTE RHEUMATISM. T. C. MACDONALD, Brit. M. J. **1**:992 (April 29) 1950.

The author has compiled figures on the number of cases and incidence per thousand patients of tonsillitis, scarlet fever, and acute rheumatism in the R. A. F. and W. A. A. F. and has reported his findings.

In the discussion he points out that tonsillitis is a "blunderbuss" term unless supported by bacterial proof. He feels that in the past too much emphasis has been placed on the value of tonsillitis with acute rheumatism, and his conclusions are very well summarized as follows: "(*a*) Tonsillitis figures are of very little use on a national basis as a warning of an imminent rise in acute rheumatism morbidity. In any case they are not available for the civil population. (*b*) Scarlet fever figures are available and it would appear that in the adult (but not aged) population there can be expected about the same number of acute rheumatism cases as there are of scarlet fever. (*c*) A rising national scarlet-fever rate should be a signal for medical

officers of health to look for the haemolytic streptococcus in epidemics of sore throats, however small these epidemics may be. (*d*) The R. A. F. and similar statistics might be of use to public health authorities, as they include non-notifiable diseases." BIRDSONG, Charlottesville, Va.

ORAL PENICILLIN IN THE PROPHYLAXIS OF STREPTOCOCCAL INFECTION AND RHEUMATIC RELAPSE. J. A. EVANS and M. B. PITT, Proc. of Roy. Soc. Med. **43**:206 (March) 1950.

A preliminary report of an investigation into the value of penicillin in the prevention of streptococcal infections among children in a home for convalescent rheumatic fever patients indicates that a daily dose of 100,000 units of penicillin given orally on an empty stomach has considerable value as a prophylactic against streptococcal infection and consequently rheumatic relapses among rheumatic children. HEARD, Monterey, Calif.

EPIDEMIC OF THRUSH OF THE SKIN WITH THE PICTURE OF ERYTHRODERMA DESQUAMATIVUM. J. B. MAYER, H. GOTZ, and GUDUN SEITZ, Ann. pædiat. **177**:213 (Oct.) 1951.

The authors observed in 40 infants the epidemic appearance of soreness and extensive intertrigo. In the further course of the illness clinical pictures appeared which showed all the transitional stages between seborrheic dermatitis, and fully developed erythroderma desquamativum (Leiner). In 35 children skin and feces were examined bacteriologically: Candida albicans was found 30 times in the feces and 17 times on the skin. The illness was therefore considered to be due to a cutaneous thrush infection. It was clinically not possible to differentiate seborrheic dermatitis from erythroderma desquamativum. Only infants up to the fifth month were involved. The epidemic died down quickly after specific treatment and strict isolation were instituted. FROM THE AUTHORS' SUMMARY.

AGGLUTINATION TITERS IN THE BLOOD OF CHILDREN WHO HAD BEEN CURED OF TYPHOID BY CHLORAMPHENICOL GIVEN ORALLY OR RECTALLY. F. TOSCANO and O. ANDREONI, Minerva pediat. **2**:378 (Aug.) 1950.

Widal tests were done on the sera of 15 children one to three months after they had recovered from typhoid or paratyphoid, during which illness they had received chloramphenicol medication either orally or rectally. The agglutination titer of the patients receiving the oral medication had fallen from 1:80 to negative. The titer in the patients receiving the rectal medication and in one patient who had received vaccine as well as oral treatment did not show this striking fall.

HIGGINS, Boston.

ABSORPTION AND ELIMINATION OF GENTISIC ACID IN NORMAL AND RHEUMATIC CHILDREN. F. TOSCANO, Minerva pediat. **2**:443 (Sept.) 1950.

A new method of analysis (Ginoulhiac and Mainardi) permits more accurate studies on the absorption and elimination of gentisic acid. From 75 to 95% of the intake was eliminated through the urine. When the sodium salt was given intravenously, most of it had been eliminated within three hours; if taken by mouth, the elimination was much slower, requiring about 24 hours.

HIGGINS, Boston.

HEREDITARY FAMILIAL ALLERGIC PREDISPOSITION IN FAVISM. G. MURANO, Pediatria **59**:479 (July) 1951.

Of 130 persons in 11 three-generation family trees, 25 (19%) had favism and 70 (54%) allergic conditions. These data indicate that favism is probably of an allergic nature. The incidence of favism fits in with Mendel's law in that there is a higher frequency of favism occurring in children of parents who are homozygous as regards the allergic factor.

HIGGINS, Boston.

MENINGEAL REACTION IN RHEUMATIC DISEASES. LELIO NASSI, Riv. clin. pediat. **47**:529 (Aug.) 1949.

Attention is called to the relative frequency of meningeal reactions during rheumatic disease, and the cases of 14 patients observed during 1948 are reported. Of these, the majority showed serous reactions: in three cases lymphocytic meningitis was observed: one patient had purulent meningitis without bacteria and with normal reducing substances and chlorides. Prognosis was consistently favorable, except for one patient who had very severe encephalitic reactions.

A thorough knowledge of these forms of rheumatic meningitis would have general interest—especially the lymphocytic types—because of the differential diagnosis which must be made with other forms of meningitis, but the author believes they also have importance because of occurring later on after a certain lapse of time since the first rheumatic attack. Referring especially to the belief that rheumatic disease is allergic—and therefore relapses would easily occur—the author states that it is probable that subacute or chronic meningeal involvement connected with rheumatic disease may take place. It is therefore necessary, bearing in mind this possibility, to effect and perfect the prophylaxis of relapses from rheumatic disease. FROM THE AUTHOR'S SUMMARY.

COMMENTS REGARDING THE USE AND MISUSE OF CHLOROMYCETIN IN TYPHOID FEVER. ANTONIO MALLOU LABRADOR, Acta pediat. españ. **8**:1213-1217 (Oct.) 1950.

Labrador, while admitting the excellent results with chloramphenicol (chloromycetin®) in typhoid fever, warns that it is not the whole answer. It must be given early in the disease if it is to prevent ulcerations and hemorrhages. If anatomical lesions exist they must be treated with hemostatic drugs, vitamins C and K, proper diet, and surgery if necessary. Chloramphenicol is an antibiotic and has no effect on already existing anatomical lesions caused by the typhoid bacillus.

For small infants, chloramphenicol may be removed from the gelatin capsule and mixed with honey or some similar vehicle immediately before administration. The use by rectum should not be tried, as, because of the very nature of the disease, the drug will be expelled before it can be used. Penicillin is often valuable in conjunction with chloramphenicol, as the child may have other organisms complicating the typhoid infection. MARY ALLEN, San Francisco.

INTESTINAL PERFORATION IN TYPHOID. CARLOS SARINANA and OSCAR NAVARRO F., Bol. méd. Hosp. inf., Mexico **7**:178 (April) 1950.

A presentation is made of 35 cases of intestinal perforation occurring with typhoid; the patients were surgically treated, with 69% recovery and 31% mortality. A comparison is made with previous statistics, and diagnosis, prognosis, and preoperative and postoperative care, with mention of the postoperative complications, are discussed. FROM THE AUTHORS' SUMMARY.

DOSAGE OF PHENOTHIAZINE FOR THE TREATMENT OF OXYURIASIS. HEINRICH EUCKER, Deutsche med. Wchnschr. **75**:361 (March 17) 1950.

It is recommended that phenothiazine tablets be given in a daily dosage of 1 to 2.1 gm. for children from one to fifteen years. These relatively large doses were well tolerated. Of 33 patients, 23 remained worm-free for longer periods. Among the 33 patients were 24 children.

AMBERG, Rochester, Minn.

ROENTGENOLOGICAL DEMONSTRATION OF ASCARIS IN CHILDREN. R. GARSCHE, Kinderärztl. Praxis **16**:369 (Nov.-Dec.) 1948.

In many cases of Ascaris lumbricoides in the intestinal tract of children, eggs cannot be found in the stools, and the eosinophilia in the blood is also not a reliable test. Garsche has studied a roentgenological method. He found that if children between 2 and 14 years old fast the night before the barium meal is offered, the Ascarides will ingest the barium and their gut can be seen roentgenographically. "Citobarium," 40 gm. mixed with 30 cc. water, is fed in the morning before breakfast—3 tablespoons to children from 2 to 6 years and 5 to 6 tablespoons to

children from 6 to 14. Under slight compression a picture is taken half an hour to an hour after the meal. The Ascarides appear in the form of curved streaks in the small intestine.

The author suggests further studies to find a barium mixture which might be taken even more avidly by the parasites.

GLASER, Chicago.

THE PROBLEM OF RHEUMATISM. V. GORLITZER VON MUNDY, Med. Klin. **46**:110 (Jan. 26) 1951.

Attention is called to the relative protection which pregnancy provides against polyarthritis.

AMBERG, Rochester, Minn.

TREATMENT OF INTESTINAL CESTODIASIS WITH ARECA NUT BY THE TRANSDUODENAL METHOD. C. OUYANG, and H. C. KAO, Chinese M. J. **69**:218 (May-June) 1951.

Decoction of Areca nut, administered through a duodenal tube, is effective against Taenia saginata and Taenia solium.

MACDONALD, Pittsburgh.

Chronic Infectious Diseases

THE DISTRIBUTION OF COCCIDIOIDOMYCOSIS IN SOUTHERN CALIFORNIA. JOHN F. KESSEL, MARJORIE BIDDLE, HAROLD A. TUCKER, and ANTOINETTE YEAMAN, California Med. **73**:317 (Oct.) 1950.

In coccidioidin skin test surveys among persons of high school age in Saugus, Canoga Park, Banning, and Palm Springs areas, the average incidence of positive reaction was 15%. Although considerably less than the 68% incidence that was reported among high school students of Kern County, it is high enough to indicate pockets of relatively high endemicity in southern California below the San Joaquin Valley.

Histoplasmin tests were performed on most of the persons tested with coccidioidin in this survey. The over-all incidence of positive reaction in the group was 7.6%. Most of the subjects with positive reaction to histoplasmin gave a history of having previously lived in some area in the central United States where histoplasmosis is known to be endemic.

A few subjects who had positive reaction to coccidioidin tests and who had lived in areas known to be endemic for coccidioidomycosis but not for histoplasmosis also had positive reaction to histoplasmin. However, the induration produced was always smaller than that caused by the coccidioidin reaction, and there was minimal confusion in interpreting the tests.

PRYOR, Redwood City, Calif.

STUDY OF ADOLESCENT CHILDREN INOCULATED WITH BCG IN EARLY INFANCY. MILTON I. LEVINE, Pediatrics **6**:853 (Dec.) 1950.

This study consists of a follow-up of 1,165 adolescent children, of whom 601 had been vaccinated with BCG during infancy. The remainder, 564, had been followed from infancy as controls.

Approximately 550 of these patients received roentgenographic examinations, the results of which were compared with roentgenograms taken during the first five years of life.

As was to have been expected, roentgenographic evidence of healed lesions of primary pulmonary tuberculosis was found much more frequently in the control group than in the group vaccinated with BCG.

Of the 298 vaccinated patients on whom roentgenograms were made, there were three with evidence of a healed pulmonary primary complex. There were also two patients with reinfection tuberculosis of the lungs. Of the 286 controls, there were 27 showing evidence of healed pulmonary primary complex. There were no cases of reinfection tuberculosis in the control group.

A study of the three vaccinated patients in whom evidence of a healed primary pulmonary complex developed suggests that in two of the cases the BCG vaccine was weak or inadequate, while in the third case the child had been exposed and was probably vaccinated in the preallergic phase of a human tuberculosis infection. In the case of the two vaccinated patients in whom

signs of a reinfection tuberculosis developed in adolescence, it is evident for the first that the allergy following a weak BCG inoculation subsided and the child received a subsequent exposure to open tuberculosis, later with the development of the reinfection tuberculosis. The second patient with reinfection tuberculosis showed evidence of having lost the tuberculin allergy approximately three years after inoculation.

As yet no evidence is forthcoming on the ability of BCG vaccine given in infancy to diminish the tuberculosis mortality rate among adolescents and young adults. There were no tuberculosis deaths in either group.

It is evident that BCG inoculation has the ability to prevent a pulmonary primary tuberculosis, at least during the period of postvaccination tuberculin allergy.

There is no evidence to indicate that BCG given in infancy is capable of preventing reinfection tuberculosis in adolescence. It is yet to be demonstrated that if the post-BCG tuberculin allergy is maintained, the reinfection type of tuberculosis will be avoided.

It would appear from this study that there is great difficulty in determining the efficacy of BCG unless some method of maintaining the potency be devised and a standard accurate means of inoculation be used.

It is suggested by this study that the BCG tuberculin sensitivity be maintained by revaccination whenever the skin reaction becomes negative. FROM THE AUTHOR'S SUMMARY.

MIDDLEBROOK-DUBOS HEMAGGLUTINATION REACTION: STUDY OF THE TEST IN CHILDREN. H. W. ANDERSON and R. V. PLATOU, Pediatrics **8**:498 (Oct.) 1951.

The Middlebrook-Dubos hemagglutination test, measuring a humoral antibody against a polysaccharide fraction of the tubercle bacillus, has been studied in 343 serum samples from 287 infants and children. The test appears to be practical and may be useful as an index to activity of tuberculous disease.

A transient rise in level of antibodies was observed in the sera of some patients after either negative or positive skin reactions were obtained with old tuberculin; this occasional provocative effect is largely dissipated within a month or two. Little or no antibody may be demonstrated in most sera from patients with either far-advanced or healing tuberculosis. An age factor makes "unexpectedly" negative serologic results in most infants and younger children apparently quite meaningless. FROM THE AUTHORS' SUMMARY.

COMPARISON OF COMBINATION THERAPY BY STREPTOMYCIN WITH THIOSEMICARBAZON OR WITH PARAAMINOSALICYLIC ACID AS WELL AS WITH TWO NEW PARAAMINOSALICYLIC ACID DERIVATIVES IN EXPERIMENTAL TUBERCULOSIS. S. MOESCHLIN and B. DEMIRAL, Schweiz. med. Wchnschr. **80**:373 (April 15) 1950.

In experiments with tuberculous guinea pigs, treatment with a combination of streptomycin and paraaminosalicylic acid proved superior to a combination of streptomycin with amithiozone (thiosemicarbazone, TB-1). The other paraaminosalicylic acid derivatives did not give better results than simple paraaminosalicylic acid. AMBERG, Rochester, Minn.

GRÖER'S METHOD OF ALLERGOMETRY IN TUBERCULOUS CHILDREN. G. LODI, Clin. pediat. **31**:280 (May) 1949.

An exact measurement of the degree of allergy to tuberculin is not easy or possible, for several reasons. Other allergens in the tuberculin may give reactions paralleling that of the tuberculin factor. Dilution may affect the response. The exact location on the skin of the test and repetition of tests certainly modify the intensity of the reaction. HIGGINS, Boston.

OUR EXPERIENCE WITH CONGENITAL SYPHILIS. PEDROZA, TARACENA, and SAINZ DE LAS TERREROS AMEZAGA, Acta pediat. españ. **8**:1191-1200 (Oct.) 1950.

The authors present the results of treatment in 236 cases of congenital syphilis at the Provincial Institute of Puericulture in Madrid.

Many of the patients treated had other lesions concomitant with syphilis, but not due to syphilis, including Oppenheim's disease, Ritter's disease, encephalitis, and Mongolism. The

others had various symptoms readily associated with syphilis, such as enlarged spleen, skin lesions, keratitis, and dry coryza.

Review of case histories showed that 33 patients were treated exclusively with mercurial ointment. Of these only 2 children survived. Poor results were also obtained with the use of various arsenicals alone. Better results were obtained with acetarsone alone than with other arsenicals.

Best results were obtained with acetarsone by mouth and penicillin. Only one death occurred in this group. The pencillin was given after nine days' treatment with acetarsone. A total of 500,000 units of penicillin was given in doses of 10,000 units every 3 hours.

The authors conclude that (1) congenital syphilis continues to be a social problem; (2) it is one of the four most frequent causes of death at the Provincial Institute of Puericulture; (3) there is great variation in symptoms in infants, some having very few and very obscure symptoms but positive serologic reactions: (4) there has been an increase in the incidence of syphilis in the last few years; (5) penicillin with arsenicals by mouth has proved to be the best treatment at the Institute.

ALLEN, San Francisco.

ATYPICAL CASE OF TUBERCULOSIS MENINGITIS WITH PULMONARY LESIONS. MATEO LARRAURI and XAVIER MATOS, Acta pediat. espan. **8**:1327 (Nov.) 1950.

The authors report a case of tuberculosis meningitis in a 6-year-old boy. Unusual aspects were an upper respiratory infection which responded to streptomycin and gave roentgenographic evidence of tuberculosis. This disease was later proved at autopsy when massive evidence of involvement in the bronchial lymph nodes and in parenchyma of the lungs was seen.

Neurological signs developed, including a hemiplegia with spastic paralysis, normal eye reflexes, and depression of the fontanel. The spinal fluid was not under pressure, but the laboratory findings were typical of meningitis. Streptomycin, administered intrathecally, did not help. The child had several epileptiform seizures, and before death a widespread purpuric rash developed. Reaction to the Mantoux test was strongly positive.

The authors include a detailed pathological study from autopsy. All types of pulmonary lesions typical of tuberculosis were found, including cavity formation and fibrous organization in the walls of the cavity, and in the lymph nodes in adjacent tissue the primary tuberculous lesions commonly associated with childhood type of tuberculosis were seen. The neurological findings were also atypical. The authors diagnosed the disease as a meningoencephalitis, and they feel it could be classified as an early generalized infection or malignant syndrome of Hubschmann.

ALLEN, San Francisco.

CONTRIBUTION TO INFANTS' TOXOPLASMA INFECTION AND CLINICAL EVALUATION OF THE SABIN-FELDMAN TEST. WERNER STROBEL, Deutsche med. Wchnschr. **76**:1433 (Nov. 16) 1951.

One of two infants died on the 25th day of life. Typical intracerebral calcifications were found upon roentgenological examination of the skull, and at autopsy the diagnosis of Toxoplasma infection was verified histologically. The other infant died on the 43d day of life. Areas of calcification in the brain were found at autopsy, and the causative agent could be demonstrated histologically in the central nervous system. The reaction of the mother's blood to the Sabin-Feldman test was markedly positive seven weeks after delivery, and the titer was 1:800; five months after delivery the titer was 1:400. The Sabin-Feldman test is done with inactivated serum. Toxoplasma loses the capacity to stain with alkaline methylene blue solution after exposure to specific antibodies in the presence of a nonspecific activator present in normal human serum and that of some animals. A series of tests was done with blood from controls and former patients who might possibly have Toxoplasma infection. The results of 13 such cases, all in infants and children, are tabulated. It is rather questionable that a single positive reaction allows a certain diagnosis of Toxoplasma infection, since the reaction can become negative rather rapidly. It may even be repeatedly positive with the result that when the micro-organism cannot be demonstrated it must be evaluated in connection with the clinical picture. For diagnosis of latent Toxoplasma infection the test must be confirmed by other serological tests.

AMBERG, Rochester, Minn.

SPINAL FLUID STUDIES: I. RELATIONSHIP OF CEREBROSPINAL FLUID AND BLOOD SUGAR LEVELS IN HEALTHY CHILDREN AND CHILDREN WITH TUBERCULOUS MENINGITIS. H. KIRCHMAIR, Ztschr. Kinderh. **66**:274, 1949.

Whereas there is a definite and constant relationship between the sugar of the cerebrospinal fluid and that of the blood in healthy children, the spinal fluid sugar amounting to an average of 62.1% of the blood sugar, this relationship is disturbed in patients with tuberculous meningitis, the average blood sugar values being higher and the spinal fluid values lower than normal.

GLASER, Jerusalem, Israel.

OBSERVATIONS OF TREATMENT EXPERIMENTS OF TUBERCULOUS MENINGITIS WITH STREPTOMYCIN. R. DUBOIS, R. LINZ, R. DELCOURT, and H. VAN WIEN, Acta pædiat. belg. **3**:5 (Pt. 1 and 2) 1949.

An entire issue is devoted to the use of streptomycin for patients with tuberculous meningitis. In the first part the authors analyze the results of various treatment methods after two years of observation and come to the conclusion that, although other methods may show good results, intramuscular and intrathecal administration of streptomycin according to a rigorous regimen gives the patient the best chance for cure.

In the second part the results of laboratory analyses are shown to demonstrate that intramuscular injections alone do not produce sufficient spinal fluid level, that intrathecal injections without intramuscular injections cause rapid disappearance of streptomycin from the spinal fluid, and that only intramuscular and intrathecal injections together produced adequate intrathecal streptomycin levels.

The third part describes the microscopic findings in patients who died after prolonged treatment.

GLASER, Jerusalem, Israel.

EARLY DIAGNOSIS OF TUBERCULOUS MENINGITIS. J. LOUTE, Acta pædiat. belg. **3**:197 (Pt. 4) 1949.

Since the introduction of streptomycin, early diagnosis of tuberculous meningitis is no longer of only academic interest. By means of a statistical table the author shows that the survival rate is greater and the sequelae rarer in patients who are treated early.

The cases of 85 patients with tuberculous meningitis are analyzed to demonstrate characteristic factors for early diagnosis. The highest incidence occurs in children in the second and third years, with a seasonal peak in March and December. Primary tuberculosis was the cause in 89% of the cases. Headaches, character changes, vomiting, and constipation were the earliest signs in older children, and convulsions occurred early in small infants. The earliest and most characteristic finding in the spinal fluid was diminution of the sugar level. The author states that a ratio of blood sugar to spinal fluid sugar above 2 occurs only in tuberculous meningitis and purulent meningeal conditions, which are easily differentiated, and he therefore finds this test the most reliable, except for the actual finding of the tuberculous bacilli.

GLASER, Jerusalem, Israel.

TOXOPLASMOSIS: SEROLOGICAL INVESTIGATION. J. WINSSER and O. MAKSTENIEKS, Nederl. tijdschr. geneesk. **93**:3630 (Oct. 22) 1949.

Sixty-three sera were examined with neutralizing Toxoplasma antibodies. The rabbit skin test and the in vitro test of Sabin were used. The neutralizing effect of serum which contains antibodies is diminished or can be lost by keeping the serum for some time at a temperature above 0 C. By adding fresh serum which contains no Toxoplasma antibodies the neutralizing effect is regained. Thus, apart from the specific antibodies, a thermolabile factor is necessary for the in vitro inhibition test. Winsser and Makstenieks simplified this test, which by this modification is made more useful in routine work. The rabbit skin test also gives better results when fresh human serum which does not contain Toxoplasma antibodies is added. The results obtained in the rabbit skin test were not consistent with those of the in vitro test in all cases.

A positive reaction to the serological tests can never serve as a proof of the diagnosis of toxoplasmosis. It can, however, be of great diagnostic value; especially if an increase in the titer can be observed during the illness.

VAN CREVELD, Amsterdam, Netherlands.

THE THERAPEUTIC VALUE OF SULFONAMIDES, ARSENIC COMPOUNDS AND ANTIMALARIA DRUGS IN EXPERIMENTAL TOXOPLASMOSIS. P. H. VAN THIEL, Nederl. tijdschr. geneesk. **93**:3818 (Nov. 5) 1949.

The results of the experimental studies by von Thiel are in accordance with those obtained by Sabin and Warren. Arsenic compounds and antimalaria drugs did not alter the course of the disease in mice which had been inoculated with Toxoplasma.

Good therapeutic results were obtained with sulfamethylpyrimidine ("percoccide"), sulfamezathine, and sulfadiazine in mice, after intracerebral inoculation with Toxoplasma. When intraperitoneal inoculation was used, no difference was observed between the effect of sulfapyridine and sulfathiazole and that of the above-mentioned chemotherapeutics. In some of the treated mice a latent infection persisted. Other mice, however, recovered completely.

Sulfamethylpyrimidine is recommended for treatment of toxoplasmosis in man. It is resorbed rapidly, is excreted rather slowly, and can be administered during a long period without toxic effect.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Blood, Heart and Blood Vessels, and Spleen

ARTERIO-VENOUS ANEURYSM OF THE FOOT FOLLOWING PLANTAR FASCIOTOMY. J. J. COUGHLIN, Northwest Med. **50**:772 (Oct.) 1951.

A case is reported of a complication following a well-known and accepted orthopedic procedure. A 10-year-old boy presented himself for treatment of recurrent bilateral clubfoot deformity. After failure of conservative therapy, it was decided that surgical treatment was indicated. A tendo Achilles lengthening and plantar fasciotomy was performed on both feet.

The patient had an uneventful convalescence, and the immediate postoperative results were considered good. Approximately eight weeks after surgery the patient returned complaining of a painful swollen area over the medial plantar surface of the right foot, immediately underlying the site of the plantar fasciotomy. After further examination it was deemed advisable to explore the region of the mass. By careful dissection a mass was isolated, and on the dorsal surface it was found to communicate proximally and distally with a vein and artery. The vessels were isolated, both proximally and distally, clamped separately, ligated, and severed, and the mass was removed. Pathological examination confirmed the impression of an arteriovenous aneurysm of a branch of the medial plantar artery and vein which were inadvertently severed at the time of the plantar fasciotomy.

DOCTOR, Seattle.

PAROXYSMAL HYPERTENSION SIMULATING PHEOCHROMOCYTOMA. NATALIE ARONSON, GERTRUDE S. STERN and SIDNEY Q. COHLAN, Pediatrics **8**:664 (Nov.) 1951.

An 8-year-old child with intermittent attacks of hypertension and vomiting has been described. The blood pressure fell dramatically in response to piperoxan hydrochloride (benzodioxan), suggesting the presence of a pheochromocytoma, but this tumor was not found after an extensive exploratory laparotomy. The episodes of hypertension and vomiting, together with the associated findings in this child of crying without tears, frequent blotching of the skin, and excessive sweating and salivating, are considered to be manifestations of a syndrome of which the cause and pathology are not well understood. A younger sibling is reported with a similar autonomic nervous-system disturbance, but the child has not yet exhibited spontaneous attacks of hypertension and vomiting.

A variety of adrenolytic and sympathicolytic agents, as well as cortisone and corticotropin (ACTH), were studied for their effects on the patient's signs and symptoms, but none of these was demonstrated to have any therapeutic value.

FROM THE AUTHORS' SUMMARY.

A NEW PROTHROMBOPENIC AGENT. ANCEL BLANSTEIN, Canad. M. A. J. **62**:470 (May) 1950.

Clinical studies on 20 patients treated with phenylindandione indicate that the drug has an effectiveness somewhere between heparin and bishydroxycoumarin (dicumarol®). Phenylinandione appears to act in half the time required by bishydroxycoumarin, and its catabolism is more rapid.

McGUIGAN, Evanston, Ill.

COOLEY'S ANAEMIA IN SINHALESE CHILDREN. C. C. DE SILVA and C. E. S. WEERATUNGE, Arch. Dis. Childhood **26**:224 (June) 1951.

Four cases of Cooley's anemia (hereditary leptocytosis) are reported from Ceylon in Sinhalese boys. In the two older boys there were marked roentgenological changes in the skull, long bones, and metacarpals and phalanges. Typical blood changes were observed.

KELLY, Milwaukee.

THE DEVELOPMENT OF MEDITERRANEAN ANAEMIA. A. H. BANTON, Arch. Dis. Childhood **26**:235 (June) 1951.

The authors have observed two cases of Mediterranean anemia (hereditary leptocytosis) hematologically since shortly after birth and have demonstrated the development of the disease in the early months of life. Possible theories for the causation and development of Mediterranean anemia are discussed.

KELLY, Milwaukee.

A CASE OF FANCONI'S ANAEMIA. W. BEAUTYMAN, Arch. Dis. Childhood **26**:238 (June) 1951.

A case of Fanconi's disease (infantile macrocytic anemia of pernicious anemia type) is described, showing pigmentation of the skin, congenital dislocation of the hips, abnormality of the left kidney, convergent strabismus, a sacrococcygeal sinus, underdevelopment, mental backwardness, and anemia with granulopenia and thrombopenia. The patient's parents were first cousins. The origin, prognosis, and treatment are discussed briefly.

KELLY, Milwaukee.

SUBACUTE BACTERIAL ENDOCARDITIS IN A YOUNG CHILD. BERNHARD LAURANCE, Arch. Dis. Childhood **26**:249 (June) 1951.

A case is reported of congenital aortic stenosis in which subacute bacterial endocarditis occurred at the age of 3 years. Although the infection was apparently cured, the child suffered embolic occlusion of the brachial and femoral arteries. Seventeen days after discharge from the hospital he died of heart failure due to an increasing aortic stenosis, as the result of fibrosis of vegetations on the aortic valve.

KELLY, Milwaukee.

ACUTE INFECTIOUS LYMPHOCYTOSIS. J. BERNARD, Pédiatrie **38**:583 (No. 6) 1949.

The author has observed a small epidemic of nine cases of this disease in the course of 18 months. The symptomatology, as well as the blood and bone marrow findings, are described and the differential diagnostic possibilities considered.

GLASER, Jerusalem, Israel.

FUNCTIONAL SYSTOLIC HEART MURMUR. G. GRANATA, Minerva pediat. **2**:533 (Nov.) 1950.

This paper discusses the characteristics of functional systolic heart murmurs as distinguished from organic murmurs.

HIGGINS, Boston.

GALLOP HEART RHYTHM IN CHILDREN. G. GRANATA, Minerva pediat. **2**:535 (Nov.) 1950.

The author believes that a dilatation of the ventricles is present in every case of gallop rhythm. The prognosis for children with gallop rhythm is not as poor as the prognosis for adults.

HIGGINS, Boston.

ACUTE HEMOLYTIC ANEMIA OF LEDERER-BRILL. A. M. CAVALLOTTI, Minerva pediat. **2**:547 (Nov.) 1950.

A 2½-year-old boy underwent two marked and nearly fatal hemolytic crises with jaundice four months apart. The second attack was associated with hepatitis and an obstructive jaundice. A diagnosis was made of Lederer-Brill type of hemolytic anemia.

HIGGINS, Boston.

CRITICAL AND EXPERIMENTAL STUDIES ON THE FUNCTIONAL RELATIONS OF THE SPLEEN TO ERYTHROPOIESIS. M. DETTORI and G. CARDINI, Riv. clin. pediat. **48**:209 (April) 1950.

The authors, after a short review of the present knowledge concerning the relation between spleen function and erythropoiesis in physiological conditions, report their comparative studies on the hematological, medullary, and peripheral changes in normal guinea pigs which have been subjected to splenectomy and made anemic through several injections of hemolytic serum and through bloodletting.

On the basis of the results obtained, they present the hypothesis, which has already been postulated by other authors, that the substances which are freed in the destruction of the erythrocytes may be able to stimulate the process of proliferation and maturity of the medullary erythropoietic series.

On the supposition that the spleen is the organ which gathers such substances together, and on the basis of their personal observations of anemic conditions in animals with splenectomy, as compared with conditions in normal animals, the authors suggest the possibility that in hemopoiesis the spleen acts to determine and regulate the hematological concentration of these substances or of substances derived from them which are able to generate morphogenetic action in the marrow.

FROM THE AUTHORS' SUMMARY.

THE BLALOCK OPERATION IN CASES OF CONGENITAL PULMONIC STENOSIS. E. DERRA, Deutsche med. Wchnschr. **75**:295 (March 3) 1950.

The results of the Blalock operation in seven patients 5½ to 8¾ years of age are given. After three to four weeks all had benefited decisively. Later the final results are to be published.

AMBERG, Rochester, Minn.

THE ADDITION OF KOLLIDON AS A SIMPLE MEANS OF OBTAINING PLASMA FOR TRANSFUSION IN CHILDREN. J. GLEISS, Ztschr. Kinderh. **66**:455, 1949.

The addition of "Kollidon" (polivinyl pyrrolidone preparation, Bayer) causes a sharp increase in the sedimentation rate of blood, thus permitting rapid and complete separation of cells from plasma of citrated blood without the use of centrifuges.

GLASER, Jerusalem, Israel.

ACUTE INFECTIOUS LYMPHOCYTOSIS. H. WEICKER and O. KNÜPFER, Ztschr. Kinderh. **66**:464, 1949.

Three cases of acute infectious lymphocytosis are described, the clinical symptoms of which were atypical. There was neither lymph-node nor spleen enlargement. The blood picture showed high white blood cell counts (40,000 to 109,000) with 73 to 81% mature small lymphocytes and a characteristically high eosinophile count (12 to 21%). The bone marrow also showed increase in lymphocytes and eosinophiles. As regards differential diagnosis, the blood and bone marrow picture in pertussis shows a characteristically low eosinophile count.

GLASER, Jerusalem, Israel.

TWO PATIENTS WITH DIFFERENT FORMS OF TRANSPOSITION OF THE GREAT VESSELS. J. H. P. JONXIS and P. J. KOOREMAN, Maandschr. kindergeneesk. **17**:136, 1949.

In a 6-month-old child with cyanosis since birth and roentgenologic signs of complete transposition of the great vessels, cardiac catheterization was done. In the right auricle the oxygen saturation of the blood was 35%, in the right ventricle also 35%, in the left auricle 87%,

and in the pulmonary vein 99%. It was not possible to catheterize one of the great vessels coming from the right ventricle. In the arterialized capillary blood an oxygen saturation of 30 to 40% was found. The general hemoglobin content of the blood (23 gm. per 100 cc.) compensated to a large extent for the low oxygen saturation, as long as the patient was resting.

A boy, 11 years old, with Taussig syndrome, was also catheterized. He had been cyanotic since birth and could walk only about 500 meters. Fluoroscopy showed that the heart was enlarged, especially the right ventricle, and the pulmonary artery was markedly dilated. The catheter went into the right auricle and right ventricle, and from there into the pulmonary artery and the aorta. In the aorta the oxygen saturation was 77%; in the pulmonary artery 85%. The pulmonary artery overrode the interventricular septum.

VAN CREVELD, Amsterdam, Netherlands.

PLASMA TRANSFUSIONS IN HEMOPHILIA. S. VAN CREVELD and M. M. PAULSSEN, Nederl. tijdschr. geneesk. **93**:2700 (Aug. 6) 1949.

The authors report their experiences with repeated plasma transfusions for hemophiliacs. The clotting time was always determined in triplicate in tubes 15 mm. wide at 37 C.

In one hemophilic child the effect of citrated plasma on the clotting time diminished after often repeated transfusions. One hour after the first transfusion the clotting time was 36 minutes; after the sixth transfusion the time was 150 minutes. A reduction of the quantity of sodium citrate had no effect. In vitro experiments showed that the citrated plasma was not diminished in its action on the clotting time. No anticoagulants in the blood of the hemophilic child could be demonstrated. A little plasma of this patient added to normal blood did not prolong the clotting time.

Transfusions of plasma, prepared in glassware, which was treated with silicone and without adding of anticoagulants diminished the time more than citrated plasma did. The large-scale preparation of "silicone" plasma was not yet possible.

In vitro experiments with heparinized plasma showed that the greatest effect on the clotting time was achieved when the dilution of normal heparinized plasma added to hemophilic blood was 1:4. The concentration of heparinized plasma in the hemophilic blood was in this case 1:40. In a higher concentration there will be a slightly adverse effect of the heparin.

Transfusions with heparinized plasma caused a greater and longer lasting diminution of the clotting time than citrated plasma did, under the condition that the quantity of heparin added to the blood was not too large. The concentration of heparin in the blood used was 1:400,000. The greatest effect in the clotting time was reached after half an hour.

In the above-mentioned child the clotting time 30 minutes after transfusion of 20 cc. of heparinized plasma was 65 minutes; after 20 cc. of citrated plasma, 180 minutes. In this case of refractoriness against citrated plasma, heparinized plasma produced the original diminution of the clotting time.

The authors indicated the sodium citrate as the cause of the refractoriness.

VAN CREVELD, Amsterdam, Netherlands.

RESULTS OF BLALOCK'S OPERATION IN A CHILD WITH FALLOT'S TETRALOGY. J. H. P. JONXIS and P. J. KOOREMAN, Nederl. tidjschr. geneesk. **93**:3956 (Nov. 19) 1949.

In a child, 5 years old, with Fallot's tetralogy, cardiac catheterization was done. As the catheter went through the foramen ovale in the left auricle and left ventricle, the oxygen saturation and the pressure could be determined in all four heart chambers as well as in the pulmonary artery and aorta. The child underwent Blalock's operation and improved markedly. After operation another cardiac catheterization was performed. The calculations revealed that 1,400 cc. of blood passed through the artificial ductus in a minute. The hemoglobin content of the blood decreased from 26 mg. per 100 cc. to 20 mg. per 100 cc., and the oxygen saturation of the arterial blood increased from 69 to 80%.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Nose, Throat, and Ear

HOARSENESS IN CHILDREN. JOHN M. LORE JR., Arch. Otolaryng. **51**:814 (June) 1950.

Children suffering from hoarseness for a considerable period should have a laryngeal examination. Often polyps or papillomas are found which can be removed. Six such cases are reported.

KIMBALL, Evanston, Ill.

THE TONSIL AFTER RADIUM IRRADIATION. ERNEST J. VAN EYCKEN, Ann. Otol., Rhin. & Laryng. **59**:648 (Sept.) 1950.

Van Eycken reports the histopathological observations on a patient upon whom tonsillectomy was performed 20 years after radium irradiation of the tonsils. The patient, a 52-year-old white woman, had been treated in 1928 by insertion of one radon seed into each tonsil for three days. It was estimated that she had received 166.8 mc.-hr. to each tonsil.

Clinical examination showed large, chronically inflamed, cryptic tonsils with injection of the anterior pillars and lingual tonsillitis. Numerous strong adhesions were encountered during tonsillectomy.

Histopathologically, the tonsils were well encapsulated, the surface epithelium showing mild hyperkeratosis. Moderate hyperplasia of the lymphoid elements was noted. Fibroplasia, with dense proliferation of scar tissue, was a prominent feature. Occasional islands of metaplastic hyaline cartilage were encountered in the fibrous tissue. Vessel walls appeared thicker than usual and showed narrowed lumina. Atrophy of the tonsils with probable irradiation sclerosis was the histopathological diagnosis.

STEELE, Kansas City, Kan. [A. M. A. Arch. Otolaryng.].

THE COMMON COLD—FACT AND FANCY. E. JAWETZ and J. C. TALBOT, California Med. **73**:379 (Nov.) 1950.

A great deal of folklore, superstition, and emotional reaction is attached to the common cold, but established objective information is quite limited. The evidence concerning etiology, epidemiology, physiology, prevention, and treatment of the common cold is briefly summarized and critically evaluated by the authors. There is a disappointing lack of real progress in any of these aspects of the common cold problem.

PRYOR, Redwood City, Calif.

USE OF HYALURONIDASE WITH LOCAL ANESTHETIC IN TONSILLECTOMY. C. J. HEINBERG, Eye, Ear, Nose & Throat Month. **30**:31 (Jan.) 1951.

Heinberg reports the use of hyaluronidase with local anesthesia in tonsillectomy. The hyaluronidase is used in a dose of 150 TR (turbidity reducing) units added to 25 cc. of 2% procaine solution combined with 5 cc. of epinephrine. Because of the more rapid and more widespread diffusion, the operative procedure is made more comfortable, the procedure may be started immediately after injection, and less of the injection solution is necessary. Healing is not retarded, and there were no untoward reactions, either local or general.

The preparation is stable indefinitely in the dry state and for about two weeks when in solution. Heinberg, however, has the solution prepared fresh for each procedure.

JENNES, Waterbury, Conn. [A. M. A. ARCH. OTOLARYNG.].

COCHLEAR SIGNS OF STREPTOMYCIN INTOXICATION. JOSEPH A. HAWKINS JR., J. Pharmacol. & Exper. Therap. **100**:38 (Sept.) 1950.

In studies on cats, the author draws the following conclusions: "A toxic action of streptomycin on the auditory system occurs in the periphery at the end organ, reducing primarily the aural microphonic response. The effect differs from that of quinine, of anoxia and of exposure to louds sounds, which depress the response of the auditory nerve fibers before they affect the microphonic."

PILCHER, Cleveland.

EVALUATION OF HEARING AIDS WORN BY SCHOOL CHILDREN. CARL H. WEAVER, J. Speech. & Hearing Disorders **16**:218 (Sept.) 1951.

An evaluation of hearing aids worn by 20 school children, ranging from the 4th to the 10th grades, resulted in the following indications: 1. The amount of gain in speech reception afforded by the aid apparently has little relation to the amount of time the aid is worn each day. 2. There is little connection between hearing loss, either aided or unaided, and the amount of time the aid is worn each day. 3. At these ages girls wear their aids significantly more than boys. 4. The amount of residual hearing seems to have no relation to the length of time the hearing aid has been worn each day.

PALMER, Wichita, Kan.

ACUTE LARYNGOTRACHEOBRONCHITIS: AN ANALYSIS OF 1,175 CASES WITH 98 TRACHEOTOMIES. A. REGINALD EVERETT, Laryngoscope **61**:113 (Feb.) 1951.

Everett reviews the cases of 1,175 patients with acute obstructive infections of the larynx, trachea, and bronchi, who were treated at the Willard Parker Hospital between 1940 and 1949. A comparison is made with similar cases occurring between 1930 and 1939. There was a marked decrease in the mortality and in operative intervention, especially intubation, in the 1940 to 1949 series. The use of the antibiotics was largely responsible for this. In addition, in the majority of patients best results were obtained by treatment with a combination of cool water vapor and oxygen.

HITSCHLER, Philadelphia [A. M. A. ARCH. OTOLARYNG.].

OBSERVATIONS IN TREATING SEVEN CASES OF CHOANAL ATRESIA BY THE TRANSPALATINE APPROACH. HAROLD OWENS, Laryngoscope **61**:304 (April) 1951.

Owens uses a transpalatine approach which is a slight modification of Ruddy's method. This method is to be preferred over the blind intranasal removal of the obstruction for many reasons, among which are these: the work is done under direct vision, the choana does not close postoperatively, and there is comparatively little postoperative care needed. The operation is described in detail and well discussed.

HITSCHLER, Philadelphia [A. M. A. ARCH. OTOLARYNG.].

FIBROUS STERNOMASTOID TUMOR OF INFANCY. JOHN GRUHN and ELLIOTT S. HURWITT, Pediatrics **8**:522 (Oct.) 1951.

A typical case of a benign fibrous sternomastoid tumor of infancy which was presented clinically without torticollis and resembled a neoplasm is reported. The natural history of this tumor is reviewed, with emphasis on the relationship to congenital muscular torticollis and the tendency toward spontaneous regression without residual deformity in four-fifths of the cases. No satisfactory explanation for the causation of these tumors is available. No unequivocal instance of malignancy at this site in infants has been reported. The recent trend toward early resection of these tumors is discussed, but the traditional practice of conservative early management with reservation of surgery for the sequelae of the tumor is recommended.

FROM THE AUTHORS' SUMMARY.

VESICULAR PHARYNGITIS: ITS RELATIONSHIP TO THE COXSACKIE GROUP OF VIRUSES. J. K. DAVID JR., DANIEL LEAVITT and BEATRICE F. HOWITT, Pediatrics **8**:672 (Nov.) 1951.

During the summers of 1949 and 1950, a number of children in Jacksonville, Fla., were observed who presented the following clinical picture. The child, usually under 5 years old, became suddenly ill with fever, malaise, and anorexia. Examination revealed vesicular or aphthous lesions on the soft palate and anterior faucial pillars. The duration of the fever was brief, usually less than 48 hours. The clinical course and the appearance of the pharyngeal lesions was strikingly uniform in these patients. A virus agent pathogenic for suckling mice was isolated from the throat secretions of 12 of 13 patients showing this clinical picture. This agent proved to be similar, but of a different serologic type, to the virus first isolated in 1948 by Daledorf and Sickles and later designated as Coxsackie virus. These results strongly suggest a causal relationship between the viral agent recovered and the clinical disease.

FROM THE AUTHORS' SUMMARY.

LARYNGEAL EMERGENCIES IN INFANTS AND CHILDREN. GEORGE S. MCREYNOLDS, South. M. J. **44**:134 (Feb.) 1951.

McReynolds discusses the differential diagnosis and treatment of congenital lesions, inflammatory lesions, neoplasms, trauma, and bulbar poliomyelitis. Frequently, especially if there is any question regarding the patency of the airway at the laryngeal level, direct laryngoscopy should be carried out. One should always be prepared to do a tracheotomy at such a time. Because laryngeal emergencies in the newborn are not common and because they are apt to occur during the night, the author, a laryngologist, suggests that pediatricians become familiar with the use of the laryngoscope.

LOGAN, Rochester, Minn.

CURE OF TWO CASES OF SUPERACUTE OTOGENOUS OSTEOMYELITIS OF THE TEMPORAL BONE IN YOUNG CHILDREN. W. SCHMIDLI, Schweiz. med. Wchnschr. **81**:461 (May 19) 1951.

The cases of two children are reported, one 6 months old and the other 2 years old. In both, otitis media, of relatively short duration and with foul-smelling discharge, suddenly gave way to very severe septicopyemic symptoms and local edema. Upon operation an unexpectedly severe destruction of bone in the mastoid and temporal bone was observed, and, in contrast, there was a rather slight destruction of the tympanic membrane and the middle ear. In spite of the absence of an epidural abscess, a superacute progressive osteomyelitis was encountered, and the diagnosis was confirmed upon histological examination. For the first patient a nonhemolytic Streptococcus was cultured. This patient was treated successfully with radical surgery and penicillin. The other responded to radical surgery, penicillin, and "elkosin" (a sulfonamide).

AMBERG, Rochester, Minn.

BROMIDE-IODIDE THERAPY IN DISEASES OF THE TONSILS OF CHILDREN. L. LANDUCCI, Clin. pediat. **33**:117 (March) 1951.

Children with diseased tonsils have been treated at a bathhouse by hydrotherapy with mineral water containing bromides and iodides. Most of the 600 thus treated showed improvement after a period of 15 days. A tonsillar test is described: The white blood cell count is made before and 60 minutes after each tonsil has received a three-minute digital massage. The reaction is regarded as positive, and indicative of infection, if the white cell count has shown an increase or decrease of more than 1,000. Of 30 children tested, all gave a positive reaction before the hydrotherapy was begun. Only eight of them gave a positive reaction at the end of the course of hydrotherapy. The author ascribes the benefit of the treatments to a general constitutional reaction rather than to local effect on the tonsils.

HIGGINS, Boston [A. M. A. ARCH. OTOLARYNG.].

HISTOPATHOLOGICAL STUDY OF TONSILS IN CHILDREN. T. PORCELLI, Minerva pediat. **2**:593 (Dec.) 1950.

From a careful study of the tonsils of 42 children the author has concluded that it is impossible anatomically to distinguish between simple hyperplasia and hyperplasia due to previous inflammation. He found no histological picture characteristic of any specific disease.

HIGGINS, Boston [A. M. A. ARCH. OTOLARYNG.].

LARYNGEAL OBSTRUCTION IN THE CHILD. JULIO C. BARANI, Arch. pediat. Uruguay **22**:688 (Sept.) 1951.

In many countries of South America maté tea is very common; kettles with boiling water are often within reach of children who innocently breathe the steam, thereby burning the mouth, pharynx, and larynx. The burn causes edema of the larynx which obstructs the larynx.

The characteristic fact is that the obstruction does not follow the accident immediately; the child first cries and then goes back to play or falls asleep. Two to six hours later the symptoms of the obstruction of the larynx appear. The evolution of the obstruction is very rapid, and in most cases the life of the child can be saved only by tracheotomy.

FROM THE AUTHOR'S SUMMARY.

INDICATIONS FOR TONSILLECTOMY AND ITS SIGNIFICANCE IN CHILDHOOD. B. LEIBER, Kinderärztl. Praxis **17**:24 (Jan.-Feb.) 1949.

Puncture of the tonsils and intracutaneous injection of autovaccines obtained from this puncture, with simultaneous control of the blood picture (eosinophiles), permits an evaluation of the state of sensitization and thus helps in finding the proper time for tonsillectomy.

GLASER, Jerusalem, Israel.

RHINOMETRIC EXAMINATIONS OF SCHOOL CHILDREN WITH ADENOID VEGETATIONS. POUL STOKSTED, Acta oto-laryng. **39**:44 (Feb.) 1951.

Nasal insufficiency of functional airflow can be determined by methods of rhinometry. This supplements objective examination. It can be used as a general screening test for school children to weed out those who require special examinations. The idea is not new, dating to the work of Brown and Claesenn in 1877. The indirect method of Zwaardemaker has been used a good deal since 1889. This method involves observing and measuring the patches of condensation on a plain mirror held just beneath the nares. All direct methods are based on a determination of the amount of air passing through the nose, the pressure in the nose during respiration, and the velocity of flow. Knowledge of any two of these factors makes the third a mathematical determinable. Respiratory curves can be kymographically recorded, thus establishing as well the needed time factor in calculations. In the author's method a fluid manometer records the ebb and flow of air, and this is transmitted to the sensitized film of an electrocardiograph. Respiratory curves are then calculated, starting from the basic formula V equals $H \times T \times D$, where V is respired volume, H is velocity of flow per second, and D is cavity lumen. T would equal time.

Results of the work have indicated that children with adenoidal obstruction have an insufficient airway; that the degree of this insufficiency is such as to cause an abnormal development in some cases of the mouth, throat, palate, and chest contour. Testing with and without the use of ephedrine and the testing of normals all tend to confirm the accuracy of the method.

LEWY, Chicago [A. M. A. ARCH. OTOLARYNG.].

Diseases of Lungs, Pleura and Mediastinum

ASTHMA IN CHILDREN: PROBLEMS IN DIAGNOSIS. CARL L. MAUSER, California Med. **73**:421 (Nov.) 1950.

Bronchial asthma in children may be difficult to diagnose. Education of the parents regarding allergic conditions, specifically bronchial asthma, is exceedingly important in order to assure satisfactory treatment and clinical results.

Chest symptoms of unexplained origin in early life should immediately arouse suspicion of allergic disease. Other causes of asthmatic symptoms must be borne in mind and excluded before a positive diagnosis of bronchial asthma is established.

Of the many factors to be considered in investigating a child with asthma, a comprehensive history is most essential. The climate to which the patient is exposed and the psychic influences must be taken into account. Physical examination, roentgenograms, and laboratory procedures should be carefully executed. Skin testing, especially with food allergens, should not be relied upon to give all the information in allergic disease. Some form of diet trial, such as elimination diets, should be used if sensitivity to food is suspected.

PRYOR, Redwood City, Calif.

STAPHYLOCOCCAL PNEUMONIA AND PYODERMA IN INFANCY. A. J. DE PANE and D. S. MCEWEN, Canad. M. A. J. **65**:439 (Nov.) 1951.

An epidemic of staphylococcal infections occurred in Winnipeg, Canada, during 1948 and 1949. Among the babies affected with pyoderma, staphylococcal pneumonia developed in six. Three of these infants died; in the others lung abscesses developed, and they recovered after protracted illness. Aureomycin appears to be the most effective drug. When it was given to all babies prophylactically for the first four days of life, the result was complete disappearance of the incidence of pustules in the babies and of breast abscesses in the mothers.

MCGUIGAN, Evanston, Ill.

A Congenital Factor in Bronchiectasis. D. J. Conway, Arch. Dis. Childhood **26**:253 (June) 1951.

The author describes four cases of bronchiectasis associated with complete transposition of the viscera. He concludes that the frequency of bronchiectasis among patients with situs inversus totalis is so much greater than in the general population as to suggest that this disease also may be due to a developmental fault.

Kelly, Milwaukee.

Alterations of Lung Tissues After Bronchography with Ioduron B. W. Vischer, Schweiz. med. Wchnschr. **81**:54 (Jan. 20) 1951.

In animal lungs as well as in those of human beings available for examination after bronchography with "ioduron B," it was shown that the actual contrast material disappears rapidly but the vehicle remains in the lungs and may produce histological changes which may be severe.

Amberg, Rochester, Minn.

Chylous and Pseudochylous Pleuritis in Children. F. Toscano, Minerva pediat. **2**:373 (Aug.) 1950.

In a child of 2½ years, after several bouts of abdominal pain and fever, a spontaneous right-sided pleural effusion developed. On thoracentesis, a milky fluid was obtained containing many cells, mostly lymphocytes, protein 3.8 gm. (albumin 1.45 gm., globulin 2.35 gm.), fat 0.48 gm., and cholesterol 370 mg. per 100 cc. Sudan III stain showed only occasional colored globules. Culture gave negative results. Four thoracenteses were done at three-day intervals, each yielding 300 to 450 cc of fluid. Recovery was complete 24 days after the onset. The author considers the case one of chylous pleurisy, in spite of the low fat content of the fluid; he favors abolishing the terms chyliform and pseudochylous pleurisy.

Higgins, Boston.

Some Neurological Complications of Virus Pneumonia in Children. C. Sticca, Minerva pediat. **2**:542 (Nov.) 1950.

Virus pneumonia is sometimes associated with meningoencephalitis or meningoradiculitis. Three such cases are reported.

Higgins, Boston.

Treatment of Empyema with Penicillin. Vicente Gimenez, Acta pediat. españ. **8**:1303-1317 (Nov.) 1950.

Gimenez reports 7 cases of children with empyema treated with penicillin. All the children recovered without surgery. X-rays and aspiration of pus confirmed the diagnosis in all cases.

The amount of penicillin used was 100,000 to 200,000 Oxford units daily, given intramuscularly, and 50,000 to 60,000, given intrapleurally every other day. The treatment was continued until aspiration yielded clear fluid. The pus disappeared rapidly and the exudate was absorbed. The general health of the children improved rapidly. The author says that even when surgery is necessary the children improve more rapidly and the mortality is less when the surgery is accompanied by the use of penicillin.

Allen, San Francisco.

Virus Etiology of Interstitial Plasma-Cellular Pneumonia in Weak Infants. K. Weisse, Ztschr. Kinderh. **67**:54, 1949.

Epidemiological studies led the author to the conclusion that interstitial plasma-cell pneumonia is a contagious disease caused by a pneumotropic virus. Animal experiments proved this point to be correct and also showed that totally atypical cases (intestinal disturbances) or the carrier state (from the environment of known cases) showed the same origin.

Glaser, Jerusalem, Israel.

Spontaneous Pneumothorax in Infants. A. Faber, Pediat. listy **6**:215 (No. 4) 1951.

The case is reported of a 3-month-old infant suffering from spontaneous pneumopyothorax which complicated the clinical course of staphylococcal septicopyemia. Recovery occurred within four weeks after administration of antibiotics.

From the Author's Summary.

TREATMENT OF PNEUMONIA WITH PENICILLIN AND SULFONAMIDES. A. LÜÜS, Svenska läk.-tidning. **45**:1791 (Sept. 17) 1948.

Since the introduction of penicillin in the treatment of acute pulmonary infections in 1945 at the hospital reported, 59 of 541 children (133 infants) with pneumonia, bronchiolitis, or empyema died. Of these, 20 died within the first 24 hours, and 26 died from other illnesses with pneumonia as a secondary disease. If these 46 cases are excluded, the total death rate was 2.4%. The death rate for pneumonia was 1.7% for all children and 6.5% for infants. The death rate for bronchiolitis was 30.8% (only infants) and for empyema 14.3%.

WALLGREN, Stockholm, Sweden.

BENIGN TERATOMA OF THE MEDIASTINUM. D. VERVAT, Maandschr. kindergeneesk. **17**:116,1949.

An 8-month-old infant who had repeatedly suffered from infections of the respiratory tract was admitted to the hospital with slight dyspnea and slight jugular and intercostal retractions and with the left side of the chest on percussion duller and showing a greater bulging than the right side. The breath sound at that area had disappeared, and the heart was displaced to the right. The roentgenogram showed a marked shift of heart and mediastinum toward the right and a large shadow with a convex lower border, which left only the left lower quadrant free. This shadow was growing rapidly. On diagnostic puncture some mucus with glial tissue was aspirated. At the operation the diagnosis of teratoma was confirmed. The smooth cystic tumor of the mediastinum was fixed to the left lobe of the thymus and could be removed easily. The extirpated benign teratoma contained mucous bronchial membrane, cartilage, glial tissue, retinal tissue, fatty tissue, and skin. After the operation, the left lung unfolded in a satisfactory manner and the heart resumed its usual place.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Gastrointestinal Tract, Liver, and Peritoneum

GASTRIC ULCER IN CHILDHOOD. M. D. INGRAM JR., Am. J. Roentgenol. **64**:765 (Nov.) 1950.

A case of gastric ulcer in an 11-year-old child is presented, and the literature, containing 33 cases, is reviewed.

The classic symptoms presented by adults with gastric ulcers are seldom seen in children. Most commonly the disease presents itself as a complication, such as hemorrhage or perforation.

The author points out the importance of subjecting all children with undiagnosed gastrointestinal symptoms to thorough roentgenologic study of the gastrointestinal tract.

HARRISON, Oak Park, Ill.

ILEOCECAL INTUSSUSCEPTION CAUSED BY LYMPHOID HYPERPLASIA. W. D. O'SULLIVAN and CHARLES G. CHILD, J. Pediat. **38**:320 (March) 1951.

The cause of intussusception in infants is poorly understood. The authors do not infer that lymphoid hyperplasia in the ileum causes all cases of ileocecal intussusception. They do report two cases in each of which lymphoid hyperplasia seemed to be the cause of the disorder.

MACDONALD, Pittsburgh.

PSEUDOMONAS AERUGINOSA INFECTION AS A COMPLICATION OF THERAPY IN PANCREATIC FIBROSIS (MUCOVISCIDOSIS). STERLING D. GARRARD, JULIUS B. RICHMOND and MARVIN M. HIRSCH, Pediatrics **8**:482 (Oct.) 1951.

Four cases of proved pancreatic fibrosis associated with the terminal bronchopulmonary manifestations in which Pseudomonas aeruginosa was the pathogenic organism have been presented. These children received prolonged antibacterial therapy varying from one to five years.

Ps. aeruginosa, or similarly resistant organisms, which ordinarily have a low pathogenicity, may become predominant and invasive as a result of prolonged antibacterial therapy. The opportunity for the emergence of resistant strains would seem to be enhanced by recent recommendations advocating continuous antibacterial therapy with a single compound. Intermittent therapy, alternating the various antibacterial agents and correlating their use with careful bacteriologic studies of the nasopharyngeal and tracheobronchial flora, would seem to be the logical

approach to treatment in the light of recent knowledge of the development of bacterial resistance. Mixtures of two antibiotics having a different mechanism of action may be more desirable than the use of a single agent.

FROM THE AUTHORS' SUMMARY.

PSEUDOCYST OF THE PANCREAS IN CHILDHOOD. JAMES MITHOEFER, Pediatrics **8**:534 (Oct.) 1951.

Because of the rarity of pseudocysts of the pancreas in childhood, the following case, the first among 101,960 admissions to the Cincinnati Children's Hospital, is reported.

A 20-month-old girl was admitted to the hospital with her abdomen hugely distended by a pseudocyst of the pancreas. Five months before the onset of abdominal enlargement, she had an illness which in the light of subsequent developments was believed to have been traumatic pancreatitis. The diagnosis of pseudocyst of the pancreas was suggested by the roentgenological demonstration of a retroperitoneal mass, which displaced the stomach upward and anteriorly, widened the duodenal loop, and displaced the ascending and transverse portions of the colon anteriorly and the left kidney inferiorly. This impression was confirmed by the demonstration of 3,200 Somogyi units of amylase enzyme in the cyst fluid and 320 units in the serum. The pseudocyst was marsupialized, and when surgically exposed, it was found to arise from the tail of the pancreas. When the patient was last seen, 10 months after operation, she was entirely asymptomatic. The abdomen was of normal size; no mass could be demonstrated by physical examination or by barium enema. The wound had been solidly healed for seven months.

A review of the literature follows. Among the many case reports, only two authenticated cases of pseudocyst occurring in childhood could be found; in each of these instances the lesion developed after severe, nonpenetrating trauma to the upper abdomen.

A classification of pancreatic cysts is given. The pathogenesis and differential diagnosis of pseudocyst are discussed.

Photographs are presented to show the patient at the time of her admission to the hospital and after her recovery. The pertinent roentgenograms are reproduced.

FROM THE AUTHOR'S SUMMARY.

CONGENITAL MEGACOLON (HIRSCHSPRUNG'S DISEASE): FOLLOW-UP ON 72 PATIENTS TREATED SURGICALLY. ORVAR SWENSON, Pediatrics **8**:542 (Oct.) 1951.

The present conception of the pathology of congenital megacolon is an absence of peristalsis in the rectosigmoid. The resultant functional obstruction in this region produces a dilatation and hypertrophy of the adjacent proximal colon. The rectosigmoid in this disease contains no Auerbach's plexus ganglion cells. As these ganglia are a part of the parasympathetic innervation from the pelvic nerves, the congenital absence of myenteric ganglion cells in the rectosigmoid in congenital megacolon probably represents a maldeveloped pelvic parasympathetic system.

Removal of the aganglionic area of bowel should cure these patients. A technic has been devised which permits removal of the diseased rectum and rectosigmoid with complete preservation of anal continence. This has now been used on 82 patients with excellent clinical results. There have been two deaths, both occurring in infants. All the surviving children have been relieved of their symptoms. The first patients were operated on four years ago. Follow-up roentgenograms reveal return of the colon to approximately normal size, 10 to 12 months after operation.

FROM THE AUTHOR'S SUMMARY.

DISCUSSION: GASTRODUODENAL HAEMORRHAGE AS A SURGICAL EMERGENCY. A. H. DOUTHWAITE AND OTHERS, Proc. Roy. Soc. Med. **43**:145 (March) 1950.

There are a few conditions, always unsuitable for surgery, which may give rise to hemorrhage and a mistaken diagnosis of peptic ulcer, namely, cirrhosis of the liver, chronic nephritis with uremia, chronic indigestion, and hemorrhage due to arterial hypertension. The only point of contention between the physician and the surgeon relates to severe hemorrhage arising from peptic ulceration. Any hemorrhage which has reduced the hemoglobin level to 50% may be regarded as severe. It is impossible to decide which of the patients who have bled severely will go on bleeding and which will stop. The importance of giving a really large transfusion has been appreciated. The solution must lie in our ability to detect earlier the persistence of bleed-

ing in patients who come in after severe hemorrhage but who do not as yet exhibit the signs of collapse. For a patient whose hemoglobin level is below 50% and who, in spite of the sedation, shows a persistently rising pulse rate and falling hemoglobin level, surgery should be done promptly. If we could treat the patient before he reaches the stage of collapse and give him a rapid transfusion before and during the operation, and continue with drip transfusion after the operation, surgical results would improve, and lives would be saved. If there is a chronic ulcer, be it in the stomach or in the duodenum, and if severe hemorrhage has occurred twice in the past, there is no doubt that surgery should be instituted in the quiescent period and the source of danger removed by one or other surgical maneuver. If the presence of a chronic ulcer be confirmed by roentgenography, a fortnight's rest in bed, correction of the anemia, and administration of a high protein diet, or, if necessary, a mixture of amino acids before surgery are recommended. There is danger of peripheral circulatory failure in using curare.

HEARD, Monterey, Calif.

CAROB-BEAN MEAL (THICKENING POWDER NESTLÉ, NESTARGEL) IN THE DIETETIC TREATMENT OF VOMITING OF INFANTS. W. HAGNAUER, Schweiz. med. Wchnschr. **79**:640 (July 16) 1949.

From the beans of ceratonia siliqua a powder is prepared containing about 41% mucin substances (pentohexosanes) but no starch. In water it swells to a pseudosolution of gelatinous consistency and high viscosity. By the addition of this powder to various kinds of milk, the milk can be thickened. Usually, 1 per cent or more of the flour was added to 100 ml. of total fluid. One advantage is that no thinning occurs on heating. Addition of the carob-bean powder proves very beneficial in certain types of vomiting in infants, but it is contraindicated when the muscles of the stomach are insufficient.

AMBERG, Rochester, Minn.

DIAGNOSIS OF VISCERAL GLYCOGENESIS BY MEANS OF MUSCULAR BIOPSY. MASSIMO ORSINI, Arch. ital. pediat. e puericolt. **12**:345, 1948.

Orsini performed the biopsy of a skeletal muscle on two children affected by glycogen-storage disease of the liver. The chemical, histological, and histochemical examinations showed changes of the glycogen-storage disease type in the muscular tissue. The changes were particularly noticeable in one of the cases.

The author suggests the introduction of muscle biopsy in the diagnosis of glycogen-storage disease of the inner organs. It is simple and harmless and perhaps as useful as liver biopsy, which is not free from serious dangers.

FROM THE AUTHOR'S SUMMARY.

ETIOPATHOGENESIS OF ACUTE INTUSSUSCEPTION IN INFANTS. G. NIERO and V. ZAFFRAGNINI, Clin. pediat. **31**:257 (May) 1949.

A theory has been evolved as to the causes usually responsible for the onset of intussusception in infants. It has been noted in the literature that intussusception is more frequent in healthy, well-nourished, and placid infants, among the nórthern (Anglo-Saxon) races, in boys, and in infants between 4 and 9 months of age. The long mesentery, the mobile cecum, the plentiful lymphatic tissue, a wider ileocecal valve, and a predominant vagotonia with increased peristalsis and intestinal spasm contribute to cause ileocecal intussusception, the commonest type of intussusception in infants.

HIGGINS, Boston.

HYPERTROPHIC STENOSIS OF THE ANTRUM PYLORICUM IN INFANTS. G. NIERO, Clin. pediat. **32**:161 (April) 1950.

The development of the pylorus is illustrated by 25 pictures of specimens from fetuses ranging in age from 3 months to birth and from infants up to 3 months old. Considerations of the embryology and physiology of the pylorus and the clinical history of cases of pyloric stenosis indicate that the condition has a neurogenic basis. It is probable that the incision of the pyloric muscle and serosa surgically is therapeutically successful by reason of the cutting of the nerve fibers to the hypertrophied musculature.

HIGGINS, Boston.

ABDOMINAL PAIN IN CHILDREN. C. SAINZ DE LOS TERREROS, Lattante **21**:425 (July) 1950.

The chief of the children's service of the Hospital de la Cruz Roja of Lisbon gives a 28-page discussion of the basis of symptoms and the differential diagnosis of abdominal pain in children.

HIGGINS, Boston.

DETERMINATION OF THE PROTHROMBIN AS A TEST OF HEPATIC FUNCTION IN THE INFANT. MARY CRISALLI and SANTE CONTE, Pediatria **55**:423, 1947.

The authors have studied the relation between prothrombin time and liver disease in children of the second and third months of infancy. Fifty children were chosen, some with primary liver disorders and some with mere hepatomegaly without apparent liver disease. It was established that while this test is sufficient in most cases (84%) to prove hepatic insufficiency, it is not exact enough to measure its extent.

The authors suggest, nevertheless, the adoption of this test in pediatrics.

FROM THE AUTHORS' SUMMARY.

GASTRIC ANALYSES IN CHILDREN. A. CARNEVALE, G. COCOZZA, and P. DE ANGELIS, Pediatria **58**:689 (Sept.) 1950.

Data are recorded of the findings in analyses of the gastric contents of 18 infants to determine acidity and chloride and pepsin.

HIGGINS, Boston.

RESULTS OBTAINED FROM ORAL ADMINISTRATION OF MATERIAL FROM LIVING CULTURES OF STREPTOMYCES GRISEUS IN TREATMENT OF INTESTINAL AND EXTRAINTESTINAL DISEASES. V. SAGGESE, Riv. clin. pediat. **47**:147 (Feb.) 1949.

The author reports the results of his current experiments with oral administration of living and whole cultures of Streptomyces griseus. Eleven children, two with enteritis, seven with enterocolitis, and two with gastroenteritis, have been treated by this method. In all cases good, and sometimes excellent, results were obtained.

These results can be explained easily as due to the antibiotic and antidotal effect of this therapeutic material locally on the diseased parts.

In addition, 14 children, 12 with pulmonary tuberculosis and two with nontuberculous pulmonary diseases, have been treated. In these cases the results have been good and sometimes excellent.

It is difficult to explain the results obtained in the therapy of these nonintestinal diseases, because of the probable loss of the antibiotic and antidotal properties.

Further research and evidence concerning this second group of diseases is needed, and the author believes that the experimental data reported should be carefully considered and studied.

FROM THE AUTHOR'S SUMMARY.

CELIAC SYNDOME: INTOLERANCE OF STARCH. RAMON MONTERO SCH. and OSCAR CORREA B., Rev. chilena pediat. **20**:481 (Nov.) 1949.

The authors report a case of mild celiac syndrome in which, besides the usual clinical findings —poor appetite; large, loose, and foul stools, and failure to gain in weight—there was a low concentration of amylase and trypsin in the duodenal juice. The patient, a boy of 3 years, responded favorably and fairly rapidly to a diet with a low content of starch, to which a commercial preparation of pancreatic enzymes was added.

Because of the diminished level of amylase and trypsin, the authors do not share Andersen's opinion that this picture should be included in the celiac disease group, and they advocated considering it as a distinct entity.

FROM THE AUTHORS' SUMMARY.

ACUTE DIARRHEA IN INFANCY AND CHILDHOOD. FELIX HURTADO and ARTURO I. ABALLI, Rev. cubana pediat. **20**:585 (Oct.) 1948.

This is a general review of the problem, particularly as it occurs in Cuba. In the same locality etiologic factors vary when patients of a different nutritional status are considered, i. e., private practice patients vs. charity hospital ward patients. Salmonella and Shigella infections

are endemic and reach epidemic proportions regularly during the summer months. The true pathogenic significance of varieties of Proteus, paracolon bacilli, and similar organisms, is still debatable. Intestinal parasites and amebae are responsible for a very small number of cases. It is difficult to decide whether virus infection may be primary or secondary.

The authors recognize parenteral diarrhea and feel that nutritional deficiency is an important factor in its etiology. They also encounter slight diarrhea due to food (no infection).

The authors emphasize the fact that well-nourished babies show a marked resistance to complications but babies seen in the infant's ward show great hydrolability.

Encephalitic manifestations are frequently seen in severe diarrhea. At autopsy marked congestion and edema of the brain are seen, with destruction and degeneration of the ganglion cells and minute hemorrhages. In only 10% could the symptoms be attributed to thrombosis of the dural sinuses.

In treatment the authors have used sulfadiazine, sulfaquanidine, succinylsulfathiazole (sulfasuxidine®), and phthalylsulfathiazole, as well as rest of the gastrointestinal tract and replacement therapy. Calcium preparations, adrenal cortical extract, and various biological preparations have not been particularly helpful, although control of recurrent diarrhea due to Salmonella or Shigella organisms by treatment with autogenous vaccine has been seen.

KEITH, Rochester, Minn.

TWO CASES OF INTESTINAL PERFORATION. PEDRO DANIEL MARTINEZ and LUIS GOMEZ OROZCO, Bol. méd. Hosp. Inf., Mexico **7**:173 (April) 1950.

A report is made of two cases of intestinal perforation at the level of the terminal ileum. In neither case was it possible to determine the cause, although the surgeon found the perforations to be similar to those seen in typhoid.

FROM THE AUTHORS' SUMMARY.

PARENTERAL INFECTIONS AND HOW THEY CAUSE NUTRITIONAL DISTURBANCES IN THE INFANT. FRANCISCO ZAMARRIEGO GARCIA, Arq. pediat. **23**:7 (Dec.) 1950.

The author gives a detailed report of the various parenteral infections of infancy and their symptoms and especially considers how the digestive system is affected. The treatment is discussed briefly.

CHRISTOPHERSON, Evanston, Ill.

CONGENITAL DIAPHRAGMATIC HERNIA. DOMINGOS DELASCIO, HERMENEGILDO MORBIN JR., and ROBERTO AIDAR AUN, Rev. ginec. e obst. **34**:9 (Oct.) 1950.

A comprehensive review is given of congenital diaphragmatic hernia, with discussions of the history, embryology, anatomy, pathological anatomy, frequency, etiology, classification, symptomatology, diagnostic signs, differential diagnosis, course, complications, prognosis, and treatment. Three cases are reported; all three patients died and on all autopsy was performed. There is an extensive bibliography.

CHRISTOPHERSON, Evanston, Ill.

SIGNIFICANCE OF THE LIVER IN THE PATHOGENESIS OF FOOD ALLERGIC GASTROINTESTINAL DISTURBANCES IN INFANCY. G. GRÜNHOLZ and H. GRÜNHOLZ, Ztschr. Kinderh. **65**:540, 1948.

Two types of antibodies are formed by the organism against foreign protein, one is physiologic, and the other exists in allergic persons, has great skin affinity, and produces histamine or histamine-like substances. The authors discuss the gastrointestinal disturbances which originate owing to this type of antigen-antibody reaction and contend that the gastrointestinal mucosa is penetrated by these antigens and that the second barrier, the liver, is also penetrated. Because of a prevalence of the sympathetic nervous system, as occurs in infections, the hepatic venous system dilates, permitting the rapid entrance of the antigen into the general circulation. This factor plus other functional damage to the liver is, in the authors' concept, the reason for the anaphylactic shock and severe intoxication in infants with an otherwise relatively harmless ailment. The authors thus feel that more attention should be paid to the protective mechanism which the liver exerts in food allergic manifestations.

GLASER, Chicago.

LIPASE TITRATION IN THE DUODENAL JUICE OF CHILDREN WITH DISTURBED FAT METABOLISM. J. GLEISS, Ztschr. Kinderh. **65**:563, 1948.

For quanitative determination of fat-splitting enzymes the author used the modified titration method of Freudenberg. Duodenal secretion was mixed with boiled cow's milk to supply the necessary fat content. The boiling of the milk effected the destruction of lipase present in cow's milk. The mixture was incubated and titrated with N/10 sodium hydroxide four times at 30-minute intervals, and the total value was expressed in cubic milliliters of N/10 sodium hydroxide minus the titration value for the cow's milk alone, which was determined first. Values between 4 and 9 cu. ml. N/10 sodium hydroxide were considered normal. Values below 4 cu. ml. must be considered as pathologic but not necessarily as indicative of a permanent pancreatic disturbance.

GLASER, Chicago.

PENICILLIN IN THE DIAGNOSIS OF THE ACUTE CONDITION WITHIN THE ABDOMEN. J. STOL, Pediat. listy **5**:166, 1950.

Stol compares two cases of primary pneumococcic peritonitis, in one of which treatment was conservative and in the other surgical. The patient treated conservatively was given 15,000 I. U. at three-hour intervals during 10 days (altogether 1,220,000 I. U.) and was hospitalized for six weeks. In the second case the operation was carried out in the first 12 hours of illness. After laparotomy and after flushing of the abdominal cavity, the child received an instillation of 100,000 I. U. of penicillin intraabdominally, a total of 360,000 I. U. of penicillin, i. e., one-fourth the dose given the patient treated conservatively, On the fifth day the patient was sent home. By comparing these facts the author comes to several important conclusions.

1. In the era of penicillin operative treatment of the primary pneumococcic peritonitis is the method most beneficial for the patient and most economical. It saves penicillin, frees the child from painful injections, and shortens very considerably the hospitalization period.

2. Stol rejects the necessity of exploratory puncture of the abdominal cavity and preliminary examination of its contents as a diagnostic help.

3. He states that the fact of utmost importance is that penicillin by its effective action has removed once and for all the old principle, that in primary peritonitis the abdomen must not be opened in the first 48 hours of the sickness. In the light of the effectiveness of penicillin the most difficult problem of differential diagnosis as between primary pneumococcic peritonitis and perforative peritonitis has become a purely academic question, for in both kinds of peritoneal inflammation immediate laparotomy is indicated.

FROM THE AUTHOR'S SUMMARY.

INTRAVENOUS IRON TREATMENT (FERRONASCIN) OF ANEMIA IN DYSPEPTIC INFANTS. HELGE GRANRUD and JACOB ÖSTER, Nord. med. **43**:468 (March 17) 1950.

Seven infants with digestive disturbance and one premature baby with anemia were treated with iron in intravenous doses of 0.1 to 1 ml. every second day in periods of 15 to 54 days. It was not possible to draw any conclusions about the effectiveness of this iron treatment. In two children toxic reactions (severe shock) occurred.

WALLGREN, Stockholm, Sweden.

INCIDENCE OF CELIAC DISEASE IN SWEDEN IN 1930-1947. KARL HENRIK CARLÉN, Svenska läk.-tidning. **45**:1399 (July 16) 1948.

Only three patients with celiac disease were treated at the Pediatric Clinic of Uppsala in 1930-1938, while 23 patients were admitted in 1939-1947. After discussion the author concludes that this increased incidence during the later period is a real one. The infants with celiac disease had been breast-fed a much shorter time than the others. Prematurity was no predisposing factor.

WALLGREN, Stockholm, Sweden.

ROLE OF SPECIFIC STRAIN OF COLI BACILLI IN EPIDEMIC GASTROENTERITIS OF INFANTS. H. BEEUWKES, A. K. A. GIJSBERTI HODENPIJL, and R. E. J. TEN SELDAM, Maandschr. kindergeneesk. **17**:195, 1949.

Bacteriological examination was done in a number of cases of infantile diarrhea. The investigation is comprised of material from four small epidemics which occurred in four towns in the eastern part of the Netherlands during the autumn of 1947.

In 37 of 44 cases of infantile diarrhea the stools contained Escherichia coli type Bray. In this group three deaths occurred. At Eindhoven this type of colon bacillus was isolated in 28 of 32 patients, all of whom were nursed in the same hospital. One of the infants had diarrhea at the time of entrance; the others had dyspepsia during their stay in the hospital. Seven children died. This type of colon bacillus gradually disappears during convalescence. The bacteria proved to be sensitive to streptomycin, as a favorable influence was seen in the patients treated with streptomycin.

Postmortem examination revealed some anomalies, which are regarded as characteristic for this disease: fat deposits in the liver, slight enlargement of the spleen, and the swollen mucous membrane of jejunum and ileum sometimes containing small hemorrhages. The mesenteric lymph nodes may be enlarged. No conclusion can be drawn about the etiological significance of E. coli type Bray in infantile diarrhea.

VAN CREVELD, Amsterdam, Netherlands.

CONGENITAL OBSTRUCTION OF THE DUODENUM AND THE UPPER PART OF THE JEJUNUM. P. P. M. NIEUWENHUIS, Maandschr. kindergeneesk. **17**:223, 1949.

Descriptions of the clinical symptoms of and the roentgenological findings in congenital obstruction of the duodenum and the upper part of the jejunum are given. It is stressed that it is not necessary to make a roentgen study with contrast medium if already after three hours much gas is collected in the duodenum and in the abdomen.

Description of four cases is given; all four patients have been treated surgically, and two have recovered.

VAN CREVELD, Amsterdam, Netherlands.

A RARE COMPLICATION OF THE OPERATION FOR OESOPHAGEAL ATRESIA. J. TEN KATE, Nederl. tijdschr. geneesk. **93**:3164 (Sept. 10) 1949.

A 5-day-old boy was operated on for esophageal atresia. At first the postoperative course was undisturbed. After four weeks the gastrostomy wound started to leak, and feeding was returned through the gastric tube. The child vomited once. Roentgenologically, a diagnosis of pyloric stenosis was confirmed. The child was operated on, and complete recovery ensued.

VAN CREVELD, Amsterdam, Netherlands.

Nervous Diseases

DIASTEMATOMYELIA. EDWARD B. D. NEUHAUSER, MARTIN H. WITTENBORG, and KLAUS DEHLINGER, Radiology **54**:659 (May) 1950.

Diastematomyelia is a congenital malformation of the neural axis characterized by a sagittal division of a segment of the spinal cord or cauda equina and usually associated with anomalous development of the vertebrae. The two lateral portions of the cord are separated by an osseous or fibrocartilaginous septum which is attached anteriorly to one or more vertebral bodies, posteriorly to the dura, and occasionally to the deformed vertebral arches as well.

As a rule no deformity is seen at birth, but later the patient fails to walk properly, or a limp develops after the child has learned to walk correctly. An abnormal gait is the most frequent initial complaint, and at that time there is usually some abnormality of one leg or both.

Roentgenograms of the spine are described. They show a fusiform widening of the interpediculate spaces over several spinal segments. In most of their cases the authors also found an osseous spur in the approximate center of the widened spinal canal. Myelograms were made preoperatively in seven patients, and all showed a filling defect of the ethyl iodophenylundecylate (pantopaque®) column at the midline bony spicule. At operation, this bony spur was found to separate the two portions of the spinal cord and to be attached to the posterior surface of the vertebral body.

AUSPACH, Chicago.

INFANTILE CEREBRAL HEMIPLEGIA: CLINICAL FEATURES AND PATHOLOGICAL ANATOMY. ROY MACKENZIE STEWART, Edinburgh M. J. **55**:488 (Aug.) 1948.

Stewart studied 112 cases of infantile hemiplegia. Almost all the patients were adults. All were patients in a home for mental defectives.

Difficult or abnormal labor and ill health of the mother during pregnancy led the list of recognizable etiological factors in the congenital cases. In the acquired group, when etiology could be determined, syphilis, encephalitis, and meningitis were most frequently found.

Paralysis occurred in the arm, leg, and face in diminishing order of severity. Atrophy was particularly manifest in the head and upper limb. X-ray examination showed an increased thickness of the skull and enlargement of the air sinuses on the affected side. Encephalography and ventriculography, apart from the rare loss of cerebral substance, showed exaggerated cortical markings and enlargement of all or part of the lateral ventricle. Dilatation of the third ventricle was less frequently seen. In a few patients the ventricular system was displaced toward the side of the lesion.

Diplegics have mental deficiency oftener and more severely than hemiplegics. However, a large proportion of hemiplegics do show some signs of such deficiency, and in this study there was a significant increase in mental deficiency in the nonparalyzed siblings. Convulsions are commoner in the hemiplegias than in the diplegias and paraplegias of infancy.

Two types of anatomical lesions were most frequent: atrophic sclerosis, either circumscribed or diffuse, and gross defects of the nature of cystic cavities or porencephaly. Hypoplasia of one cerebral hemisphere was seen in three cases without other local pathological changes. It appears from the work of others that the postcentral cortical area has a great influence on the growth of the opposite side of the body. Large lesions in the central region of the hemisphere seem to be associated with severer peripheral atrophy. Finally, the pathogenesis of hemiplegias seen in children previously in good health is an unsettled problem.

MACDONALD, Pittsburgh.

NEUROBLASTOMA SYMPATHETICUM: A SERIES OF EIGHT CASES. H. J. PASH, Clin. Rep. Adelaide Children's Hosp. **1**:150 (Nov.) 1948.

A brief discussion of neuroblastomas is presented with eight short case reports. One case presented a typical clinical picture with apparent transition to a benign ganglioneuroma.

MCGUIGAN, Evanston, Ill.

PSYCHOMOTOR INSTABILITY OF CEREBELLAR ORIGIN. P. F. GIRARD, L. THEVENIN, and R. LACHANAT, Pédiatrie **38**:9 (No. 1) 1949.

The authors describe four cases of psychomotor instability caused by lesions affecting the cerebellum as demonstrated by air encephalograms.

GLASER, Jerusalem, Israel.

MENINGITIS DUE TO B. COLI TREATED AND CURED WITH STREPTOMYCIN. SOLON VERAS and A. MYSTAKIDIS, Pédiatrie **38**:209 (No. 3) 1949.

A 2½-year-old child was affected by meningitis due to Escherichia (Bacillus) coli. The child was treated for 18 days with 100 mg. streptomycin intramuscularly every three hours and 100 mg. intrathecally every day, that it, 900 mg. daily. There was complete recovery.

GLASER, Jerusalem, Israel.

SUBDURAL HEMATOMA IN AN OLDER CHILD. P. JOSSERAND and R. ANNINO, Pédiatrie **38**:245 (No. 3) 1949.

A subdural hematoma was found in a 10-year-old child who presented signs and symptoms of meningitis. The child died two hours after surgical removal of the hematoma. There was no previous history of cranial trauma.

GLASER, Jerusalem, Israel.

HYPOPOTASSEMIC CONDITION FOLLOWING MENINGOENCEPHALITIS. L. BARTA, Ann. pædiat. **175**:343 (Oct.) 1950.

In a patient in whom hypertrichiasis developed following meningitis, constant hypopotassemia was observed. This symptom was interpreted as a sign of cortical hyperfunction. The administration of desoxycorticosterone acetate (percorten®) resulted in polyuria and in loss

of weight. These phenomena also indicated a hyperfunction of the adrenal cortex. It is assumed that hypertrichiasis which appears after tuberculous meningitis in which streptomycin has been used develops in consequence of a hypertrophy of the adrenal cortex due to dysfunction of the diencephalon, rather than as a side-effect of streptomycin.

FROM THE AUTHOR'S SUMMARY.

DIENCEPHALON SYNDROME WITH HYPERTONIA IN ENCEPHALITIS. L. BARTA, Ann. pædiat. **176**:93 (Feb.) 1951.

Signs of a vegetative neurosis (erythredema polyneuropathy) were present in a boy aged 8, the most marked sign being hypertension. The blood cholesterol was raised. The albuminocytological dissociation in the cerebrospinal fluid made the author suspect that the signs of vegetative neurosis were produced by a neurotropic virus. Reviewing some earlier cases in which hypertension was present without signs of disturbance in kidney circulation, he discusses the possibility that in this case also damage to the diencephalon was responsible.

FROM THE AUTHOR'S SUMMARY.

X-RAY THERAPY IN CHRONIC HYDROCEPHALUS IN INFANTS. A. LEONE, Ann. ital. pediat. **2**:193 (June) 1949.

The author, after a clear review of the complicated and difficult question of children's chronic hydrocephalus, reports a study made during 20 years in the Children's Hospital of the University of Cagliari.

Of 116 children observed (0.87% of all the patients), and 11 children treated in the outpatient clinic, 67 received x-ray therapy. The results were 63% of the patients without inflammation but with an augmented production of the cerebrospinal fluid, 100% of the patients with hydrocephalus from Meningococcus meningitis, 50% of the patients with meningitis from other micro-organisms, and 40% of the patients with hydrocephalus resembling encephalomeningitis, recovered.

Discussing the possible mechanism of the action, the author thinks that deep x-ray therapy can lessen the production of cerebrospinal fluid by influencing not only the choroid plexus, but also the vegetative nervous system through the diencephalic centers. This is in accord with Francioni's opinion.

FROM THE AUTHOR'S SUMMARY.

FACIAL PARALYSIS IN CHILDREN. R. CORDA, Ann. ital. pediat. **3**:367 (Aug.) 1950.

According to the author, facial paralysis of rheumatic infection is rather rare among children. This infection has occurred in only 4.8% of patients in the Children's Hospital of Cagliari; the others were nearly all poliomyelitic. The author gives the clinical symptoms in these two forms.

FROM THE AUTHOR'S SUMMARY.

FREQUENCY AND PATHOGENESIS OF GUILLAIN-BARRÉ-STROHL SYNDROME IN CHILDREN. G. GOMIRATO and U. VIGNOLO-LUTATI, Minerva pediat. **2**:525 (Nov.) 1950.

A child 2½ years old presented the typical picture of Guillain-Barré-Strohl syndrome of meningoradiculoneuritis, having a normal blood cell count but a high globulin content in the spinal fluid. This condition is probably due to a virus, and is much more rare than its counterpart, acute poliomyelitis. The different meningeal reactions in the two conditions may be explained on the basis that in radiculoneuritis the inflammation is anergic in type and in poliomyelitis the inflammation is allergic.

HIGGINS, Boston.

DIAGNOSTIC MEASURES IN SPASMOPHILIC DIATHESES AND THEIR CLINICAL VALUE. W. TANGHERONI and E. BOTTONE, Riv. clin. pediat. **48**:497 (Aug.) 1950.

Infants were given a test for hypocalcemia, by means of estradiol benzoate, and the forced respiration test, which had already been proposed as a diagnostic means in the tetany of adults. The study of chronaxy has also been associated with the biochemical inquiry on the calcium-

phosphorus metabolism. The clinical value of these tests, guided by pathological and normal cases, is discussed, and their use for the recognition of the diathesis of tetany is proposed.

The authors also trace the essential outlines of this diathesis, in which there is a predisposition to pathological manifestations and in which pathogenetic treatment might, therefore, be beneficial.

FROM THE AUTHORS' SUMMARY.

SUBDURAL ABSCESS. LATORRE B. MARIANO, Rev. chilena de pediat. **22**:386 (Sept.) 1951.

The etiology and pathogenesis of subdural empyema are briefly reviewed. The diagnostic difficulties encountered and the clinical history of a 22-month-old infant with this condition are described. In this case, the diagnosis was made during the performance of a ventriculography. During the course of the disease disparity between the presence of symptoms indicating severe intracranial hypertension and the absence of focal cerebral symptoms was noticeable.

FROM THE AUTHOR'S SUMMARY.

PURULENT MENINGITIS DUE TO MICROCOCCUS CATARRHALIS. BIECHTELER, Med. Klin. **45**:539 (April 28) 1950.

Twenty-two days after skull fracture an 11-year-old boy became ill with meningeal symptoms. The cerebrospinal fluid pressure was increased, the fluid was turbid, with 906/3 cells. The meningitis yielded relatively rapidly to penicillin treatment given in part intraspinally. From the cerebrospinal fluid Micrococcus catarrhalis was obtained in pure culture.

AMBERG, Rochester, Minn.

CASE OF BLASTOMYCES MENINGITIS. P. NALENZ and W. BOESE, Med. Klin. **46**:1130 (Oct. 26) 1951.

A boy one year of age was admitted to the hospital with suspected ileus. In the mouth a thick layer of thrush-like encrustations was seen. In the serous purulent material obtained on paracentesis of the ear drum, in material from the mouth, and in the cerebrospinal fluid, very numerous Blastomycetes were seen. In cultures mainly varieties of Torulopsis colliculosa and, to a less extent, Candida pulcherrima were found. The infection proved resistant to penicillin and sulfonamides but yielded to streptomycin.

AMBERG, Rochester, Minn.

CONTRIBUTORY DATA CONCERNING THE DECREASE OF SUGAR IN THE CEREBROSPINAL FLUID IN MENINGITIS. F. VARGA and C. KUN, Paediat. danub. **6**:9 (July) 1949.

In various cases of meningitis, the factors responsible for the decrease of the sugar level in the cerebrospinal fluid have been investigated. On incubating the fluid at 37 C. for several days, the authors found that the sugar content remained unchanged in cases of lymphocytic meningitis and in the purulent fluid in cases of meningococcal, staphylococcal, streptococcal, and pneumococcal meningitis.

The sugar content of the fluid obtained from patients with tuberculous meningitis remained unchanged for more than one month. Artificial infection with air bacteria or coli organisms resulted in a rapid decrease of the sugar level of the fluid. No correlation was found between the reduction of the fluid sugar level and the blood sugar.

Thus it can be stated that neither the leucocytes nor the bacteria found as pathogenic agents in meningitis are responsible for the diminished sugar values. The low sugar level occurring in tuberculous meningitis and idiocy could be enormously raised by the intraspinal administration of 10 ml. of a 20% dextrose solution, but the low initial values are regained within a few hours. From this phenomenon, the authors infer that the reduction of sugar in the cerebrospinal fluid is likely to be due to a nervous mechanism.

FROM THE AUTHORS' SUMMARY.

News and Comment

GENERAL NEWS

Specialists Certified by American Board of Pediatrics, Inc.—The following physicians were certified as specialists by the American Board of Pediatrics, Inc., after examinations held in New York, Feb. 1, 2, and 3, 1952:

Alperin, Leonard Jack, Cleveland.
Baehren, Paul F., Dayton, Ohio.
Bashe, Winslow Jerome, Jr., Philadelphia.
Bernardin, Ronald M., Haddonfield, N. J.
Blackmore, Betsy Snyder, Columbus, Ohio.
Bladen, George, Syracuse, N. Y.
Botstein, Anne, New York.
Calcagno, Philip Louis, Buffalo.
Cartland, John E., Hartford, Conn.
Cheung, Mung Wah, New York.
Chuan-Ying, Yang, New York.
Colburn, Russell Fitch, Stamford, Conn.
Coombs, Harrison S., New Canaan, Conn.
Crawford, Oliver Wilson, Chicago.
Davidow, Sidney L., Youngstown, Ohio.
Davis, R. Wendell, Ithaca, N. Y.
Deacon, Crosby Ball, Hamilton, Ont., Canada.
Deen, Oliver Francis, Jr., Tampa, Fla.
Dehn, Helmut Max, Berea, Ohio.
Desmond, Murdina MacFarquhar, Houston, Texas.
Dresner, Evelyn Edith, Ridgefield Park, N. J.
Edwards, Harry McCreary, Ocala, Fla.
Falcone, Joseph W., Brooklyn.
Farley, William J., Newark, N. J.
Fichter, Elaine G., New Orleans
Fletcher, Thomas F., Jr., 6600 Medical Group, % Postmaster, New York.
Ford, John R., Gloversville, N. Y.
Gaffney, Paul C., Pittsburgh.
Gilbert, Michael L., Great Neck, L. I., N. Y.
Gilder, Rodman, Jr., New York.
Githens, John Horace, Jr., Denver.
Goldberg, David, Park Forest, Ill.
Gordon, H. Samson, Chicago.
Gordon, Milton, Huntington, L. I., N. Y.
Greenfield, Theodore B., Brockton, Mass.
Gregg, Grace Stollar, Pittsburgh.
Harvey, Norman Albert, Glens Falls, N. Y.
High, Robert H., Philadephia.
Hoffman, Sumner H., Needham, Mass.
Imperiale, Louis E., Brooklyn.
Johnson, Ethel L., Flushing, N. Y.
Joos, Howard A., Rochester, N. Y.
King, William Everett, Middle Village, L. I., N. Y.
Kingsland, Lawrence C., Jr., Camp Lejeune, N. C.
Leonard, Henry, Kew Gardens, N. Y.
Lione, James G., Jackson Heights, N. Y.
McLean, Donald Emerson, Winchester, Mass.
Meltzer, Richard S., Rochester, N. Y.
Miller, Gerald, Rochester, N. Y.
Miller, Joseph B., Mobile, Ala.
Miller, Ruth Avers, Valley Stream, N. Y.
Mitty, Virginia Connolly, New York.
Nathan, Abraham, Brooklyn.
Nolke, Anthony C., Detroit.
O'Connell, W. Frederick, Rochester, N. Y.
Patrick, Albert L., Staten Island, N. Y.
Pfeffer, William, Jr., Boston.
Porter, George Edward, Pittsfield, Mass.
Prugh, Dane G., Boston.
Ripp, John Alfred, Westbury, N. Y.
Roody, Peter, New Hyde Park, L. I., N. Y.
Rosenthal, Ira Maurice, Brooklyn.
Schaffran, Morton, Aurora, Ill.
Schwartz, Jack J., East Orange, N. J.
Shoob, Milton Philip, Bayside, L. I., N. Y.
Smith, William Henry, Katonah, N. Y.
Solomons, Gerald, Providence, R. I.
Steinheimer, Mary Elizabeth, Ft. George G. Meade, Md.
Stilson, Carter, New Haven, Conn.
Sweet, Avron Y., New York.
Toch, Rudolf, 20th Station Hospital, % Postmaster, New York.
Tyler, Louis Ira, Jr., Baton Rouge, La.
Vogt, Frank Conrad, Brooklyn.
Vollmer, Hermann, New York.
Watchko, John, Indiana, Pa.
Watson, Walter Brown, Brooklyn.
Wilking, Leo F. J., Jr., New York.
Wilking, Virginia Nichols, New York.
Wiseman, William Martin, Brooklyn.

ANNOUNCEMENTS

Memorial Center for Cancer and Allied Diseases Announces Course.—The Pediatric Department of the Memorial Center for Cancer and Allied Diseases announces a comprehensive three-day course for pediatricians, general practitioners, and health officers on May, 14, 15, and 16, 1952. Current developments and established methods in diagnosis, differential diagnosis, and management of benign and malignant tumors, Hodgkin's disease, leukemia, and reticuloendothelioses in childhood are included. Content of the course is as follows: demonstrations; examinations of children in pediatric, surgical, chemotherapy, and radiotherapy clinics; ward rounds and seminars under the direction of the staffs of Memorial Hospital and Sloan-Kettering Institute for Cancer Research. The fee is $35.00. Requests for further information should be addressed to Director of Pediatric Service, Memorial Center for Cancer and Allied Diseases, 444 E. 68th St., New York.

Summer Camp for Diabetic Children.—For the fourth season a summer camp for diabetic children will be opened under the auspices of The Chicago Diabetic Association, Inc., from July 1 to July 22, 1952, at Holiday Home, Lake Geneva, Wis.

Postgraduate Course in Pediatrics.—A two weeks' postgraduate course in general pediatrics will be given at Burnham Memorial Hospital for Children, Massachusetts General Hospital, Boston, under the auspices of the Harvard Medical School and direction of Dr. Allan M. Butler, Professor of Pediatrics, June 9 through June 21, 1952. Clinics providing a broad review of pediatrics, emphasizing the new advances, will be given daily from 9:00 a. m. to 4:30 p. m. by selected authorities from the Pediatric and Specialty Services of Massachusetts General Hospital and from the Ophthalmological and Otolaryngological Services of the Massachusetts Eye and Ear Infirmary and the Children's Medical Center, Boston.

For further information write Assistant Dean, Courses for Graduates, Harvard Medical School, 25 Shattuck St., Boston.

Books

The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence. By Lawson Wilkins, M.D. Price, $13.00. Pp. 384, with numerous illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1950.

For the past 20 years textbooks of endocrinology have been available, all of them written from the point of view of the physician dealing primarily with adults. With a few exceptions, no subject in these works has been dealt with to the complete satisfaction of the pediatrician, who has been forced to maintain his private bibliography of original articles for reference when confronted with endocrine disorders in his young patients. It was therefore with mounting pleasure and satisfaction that the reviewer read farther and farther into the present volume, for here at last is a truly pediatric textbook of endocrinology.

It is immediately apparent that the author is first and foremost a clinician. The research and laboratory aspects are not neglected, but they are always considered as aids to the understanding or diagnosis of a clinical condition.

The entire approach is original, with emphasis throughout being placed on the influence of the endocrine glands upon somatic and sexual growth and development.

Each subject is approached from the standpoint of the diagnostician faced with the problem of deciding whether a particular symptom complex represents a congenital anomaly or a constitutional variation in the pattern of development or is due to an endocrine disorder which may be helped by treatment. The reader is frequently referred to other texts or articles for complete discussions of matters which, in order to preserve the admirable brevity of the text, must here be only briefly summarized. The writer is at considerable pains to discourage unnecessary treatment with endocrine preparations and at all times is conservative in his recommendations.

Even without the text the book would still be a bargain because of the tremendous amount of helpful information condensed into the numerous charts and figures. The pages of serial pictures of patients, along with thumbnail histories, are of great interest because of their clarity and the lesson taught by each. The excellently selected references at the end of each chapter are likewise invaluable.

Beautifully printed on large pages suitable for the uncrowded reproduction of the illustrations, this is virtually an essential addition to the pediatrician's bookshelf.

Health Education in the Schools. By the Massachusetts Department of Education and the Massachusetts Department of Public Health. Price, not given. Pp. 91, with 8 charts. Boston, 1949.

This excellent school health manual, which is equally helpful to public health and school personnel, states specifically the fundamental principles, policies and functions essential to a good school health program. It describes a program beginning with the complex postnatal influence on the preschool child and extending throughout his high school days. Qualifications of personnel, descriptions of equipment and an outline of program procedures, with particular reference to the exceptional child, are all included in this concisely written compendium.

An outline of the scope of this manual includes the following topics: school health services, school health programs, personnel qualifications, personnel functions and duties, first aid, health education, dental health, mental health, environment, services offered by the Massachusetts Department of Education and the Massachusetts Department of Public Health, general laws relating to school hygiene and record forms. A form for the evaluation of school health, which could serve as a measuring device for any school health program, is given at the close of the manual.

It is an impressive formulation of a job well done through excellent cooperation and team work by professional workers in the fields of both education and public health.

Language for the Preschool Deaf Child. By Grace Harris Lassman. Price, $5.50. Pp. 263, with numerous pictures and charts. Grune & Stratton, Inc., 381 4th Ave., New York 16, 1950.

This small volume will prove quite valuable to those who need guidance in the care of the preschool deaf child. It gives sound advice for parents, teachers, and doctors in the proper adjustments not only of the child but also of parents and associates in preventing conflicts and frustrations. It emphasizes especially the important part parents play, from the beginning, in developing attitudes that overcome the handicap and lead to happy, useful living. Here are answered most of the questions anxious parents ask as to what they can and should do, and most will be surprised that their part is so important and that it can be so satisfying. Information is given for future training if that is needed.

Helpful techniques for parents and teachers, such as for lipreading and training of the senses, are described understandably in some detail.

The book is well written, with sympathy and good psychologic balance. Format, paper, and printing are excellent, and illustrations are numerous and informative. It is recommended for every parent of a young child with important hearing impairment. Physicians seeing such patients will find it well worth reading.

Human Milk: Wartime Studies of Certain Vitamins and Other Constituents. By S. H. Kon and E. H. Mawson. Price, 4 shillings. Pp. 188, with 95 tables. His Majesty's Stationery Office, York House, Kingsway, London, W.C. 2, England, 1950.

The components of human milk which are investigated in this report are the fats and the nonfat solids, vitamin A and the carotenoids, vitamins C, D, B_1, and riboflavin. The samples, except when otherwise stated for special purposes, consisted of the whole milking from one breast four to six hours after it was previously emptied. The details of collection and analysis are given, and the tables giving results state the number of samples used for each mean value.

The main study was on the milk of mothers from two areas: one, a prosperous country town, Reading, and the other, a crowded part of London, Shoreditch, during the war years, 1942 to 1945. Over 2,000 samples were used. The results were analyzed in a number of different ways, for example: the fat content in relation to stage of lactation and the vitamin content in relation to giving special vitamin supplements. No marked differences were found between the milk of the women of the two groups. Neither supplied milk meeting the standards for infant requirements suggested by the United States National Research Council (1945) in respect to vitamins A, D, B_1, or riboflavin, but it is suggested that this may be too high in the case of thiamine and riboflavin, and of course vitamin A and D needs are usually met by the addition of cod liver oil. Both groups supplied adequately vitamin C. The results of surveys on the diets of Shoreditch women in comparison to the League of Nations (Magee 1943) and the United States National Research Council (1945) standards for lactating women are given. The diet rarely met these standards in all respects.

A comparison is also given between the milk of some of the Shoreditch women and that of a group of Glasgow women. It was difficult to find women in Glasgow still lactating satisfactorily in the fourth to fifth week after the birth of the child. This suggests that the quantity of milk is more easily affected by undernutrition than the quality.

Another study of the composition of milk throughout an individual lactation of 33 weeks is presented. The mother's diet was carefully supervised, and it was found that with the use of specially allowed supplements for lactating women, a fairly satisfactory diet could be provided even in wartime. Here, also, the effect of varying vitamin supplements was studied.

The object of the whole investigation, as stated, was to determine how far the content of certain vitamins and other substances in human milk reflects the state of nutrition of the mother with regard to these factors and to study the influence of wartime dietary changes. Unfortunately, the nutrition of the mothers was noted only in a most general way or by a comparison of their diets to suggested standards. Nevertheless, the large amount of data presented is of value in itself and will probably prove of basic value for further comparative studies.

Schizophrenic Art: It's Meaning in Psychotherapy. By Margaret Naumberg. Price, $10.00. Pp. 247, with 63 illustrations and 8 color plates. Grune & Stratton, Inc., New York, 1950.

This is a sound book, enjoyable to read, in which the author defines a new approach to psychotherapy and makes a worth-while additional contribution to the study of schizophrenia. The introduction gives an excellent review of psychiatric literature over the past 75 years, pertinent to psychotic and neurotic art. This begins with the descriptive studies of Simon and Lombroso of the art of the "insane," progresses through the experimental drawing tests developed by Mohr, Prinzhorn's esthetic approach, and the early explorations of art by psychoanalysis in the writings of Schilder, Pfister, Pfeifer, and others. Finally the author compares Freud's concept of symbolism with that of Jung and examines the difference between "archaic heritage" of the former and "collective unconscious" of the latter, in order to evaluate the influence of these two concepts on the use of spontaneous art in the process of therapy.

The spontaneous art expressions, in the form of paintings and sculpture, created by two adolescent schizophrenic girls form the basis of the clinical studies. The psychodynamics are clearly delineated as the cases unfold. With the patients undergoing therapy, the art productions change from archaic, fragmented stereotypes to more integrated, nonschizophrenic forms of art. Through this expression the patients are able to release their long-repressed conflicts and anxieties. As insight increases they are able to identify themselves with their creations and avoid excessive dependence upon the therapist. The author stresses the importance of spontaneous art as a mode of therapy for the patients' capacity to understand their own symbols rather than depending upon the therapist's ability to interpret their meaning.

Pediatric X-Ray Diagnosis. By John Caffey, M.D. Second edition. Price, $22.50. Pp. 862 with 1039 illustrations, The Year Book Publishers, Inc., Chicago, 1950.

Dr. Caffey's book is the only text in English on this subject to appear since 1910. The first edition was successful, and the second edition published five years later incorporates a large amount of newer material. With the addition of 385 figures the number of illustrations has been increased by over 50%. Many sections have been rewritten and expanded. Among them are the sections on primary pulmonary tuberculosis, thymus and sudden death, congenital malformations of the heart, congenital obstructions of the alimentary tract, normal variants in long bones, and cerebral toxoplasmosis. New sections have been added on pulmonary histoplasmosis, meconium peritonitis and ileus, cortical defects in tubular bones, infantile cortical hyperostosis, hypervitaminosis A, and fetal aspiration and ingestion, and there are new tables for skeletal maturation and prenatal depressions of the skull, as well as many others.

Normal structures and their normal variants are stressed, in order to give the reader a better means of evaluating pathological shadows. The reproductions of roentgenograms are outstanding in their clarity and have been carefully chosen to illustrate the disease under discussion.

The book is beautifully printed and bound, and the illustrations are uniformly excellent. Roentgenologist and pediatrician alike can profit greatly by frequent and intensive study of this book. It is comprehensive, authoritative, and scholarly.

CORRECTION

The place and time of the annual meeting of the American Pediatric Society is Hotel Chamberlin, Old Point Comfort, Va., May 7, 8 and 9, rather than Hotel Chamberlain, Old Point Comfort, Va., May 6, 7 and 8, 1952, as stated in the Directory of Pediatric Societies in the January issue, page 144. The Society for Pediatric Research will meet in the same place on May 5 and 6, 1952.

Directory of Pediatric Societies *

INTERNATIONAL

International Pediatric Association

President: Dr. Felix Hurtado, Habana, Cuba.
Secretary: Dr. Guido Fanconi, Kinderklinik, Zurich, Switzerland.
Next Meeting (Seventh International Congress of Pediatrics): Habana, Cuba, 1953.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

Pan-American Congress of Pediatrics

President: Dr. Conrado Pelfort, Montevideo, Uruguay.

International Union for Child Welfare

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 43 Quay Wilson, Geneva, Switzerland.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriquez Peña.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179.

Associação Paulista de Medicina, Secçaõ de Pediatrias

President: Dr. Armando de Arruda Sampaio.
First Secretary: Dr. Luis Fontoura y Lauro Machado Oliveira, Caixa Postal 2103, São Paulo, Brazil.
Second Secretary: Dr. Luiz Augusto Monteiro Toledo, Av. Brigadeiro Luiz Antonio 393, 1° Andar, São Paulo, Brazil.
Time: Twelfth of every month, 8:30 p. m.

Asociación Pediatrica de Guatemala

President: Dr. Carlos Manuel Monzon, Plazuela Santuario de Guadalupe, Guadalupe.
Secretary: Dr. Maria Isabel Escobar, Casa del Niño no. 1, Guadalupe.

Australian Paediatric Association

President: Dr. H. Douglas Stephens.
Secretary-Treasurer: Prof. Lorimer Dods, The Institute of Child Health, Royal Alexandra Hospital for Children, Camperdown, N. S. W., Australia.
Time: One meeting a year.

British Paediatric Society

President: Dr. A. G. Maitland-Jones.
Secretary: Dr. Alan Moncrieff, Institute of Child Health, Hospital for Sick Children, Great Ormond St., London W. C. 1.
Place: Windermere.

Bulgarian Pediatric Society

President: Dr. L. Ratchew, University Children's Hospital, Wassil Kolarow 46, Sofia.

Chinese Pediatric Society

President: Dr. Fu-tang Chu, Peking Children's Hospital, Peking, China.
Secretary: Dr. Hua-kang Chow, Peking Union Medical College, Peking, China.
Time: Concurrent with the annual conference of the Chinese Medical Association.

Confederación Sudamericana de Sociedades de Pediatría

President: Dr. Conrado Pelfort, Montevideo, Uruguay.
Secretary: Dr. José M. Portillo.
Headquarters: Avenida Agraciada 1464 (Piso 13), Montevideo, Uruguay.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Czechoslovak Pediatric Society

Chairman: Prof. Dr. Jiri Brdlík, 2 Sokolská, Prague II.

Danish Pediatric Society

Chairman: Prof. Preben Plum.
Secretary: Dr. Elin Fog, Søllingsvey 17, Charlottenlund, Denmark.

German Pediatric Society

President: Dr. H. Opitz, Heidelberg.
Secretary: Dr. Jochims, Lübeck.

Hellenic Society of Pediatrics

President: Dr. C. Saroglou, Patission St. 74, Athens, Greece.
Secretary-General: Dr. S. Charocopos, Alexandras Ave. 1, Athens, Greece.

Indian Pediatric Society

President: Dr. K. C. Chaudhuri, Dept. of Pediatrics, Medical College, Calcutta.
Secretary: Dr. S. P. Bhattacharjee, Dept. of Pediatrics, Medical College, Calcutta.
Place: Office of the Indian Medical Association, 67 Dharamtala St., Calcutta 13.
Time: Every second and fourth Wednesday of each month.

Medical Association of Israel, National Pediatric Society

President: Prof. L. F. Meyer, 29 Idelson St., Tel-Aviv, Israel.
Secretary: Dr. W. Hirsch, Hadassah Municipal Hospital, Tel-Aviv, Israel.

Nederlandsche Vereeninging voor Kindergeneeskunde

President: Dr. G. J. Huët, 39 Brediusweg, Bussum.
Secretary: Dr. H. Reerkink, % Sophia Kinderziekenhuis, 160 Gordelweg, Rotterdam.
Place: Children's Hospital or Pediatric Clinic. Time: Three times a year.

Norwegian Pediatric Society

President: Dr. Arne Njå, Oslo.
Secretary-Treasurer: Dr. Helge Granrud, Abbedikollen 38, Bestun, Oslo.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

See Medical Association of Israel, National Pediatric Society.

Pediatric Society of New Zealand

President: Dr. Lawrence Ludbrook, The Lister Building, Victoria Street East, Auckland.

Royal Society of Medicine, Section of Paediatrics

President: Dr. A. G. Watkins.
Secretary: Dr. Ronald Mac Keith; Dr. J. J. Kempton.
Place: 1 Wimpole St., London W. 1. Time: Fourth Friday of each month, 4:15 p. m.

Schweizerische Gesellschaft für Pädiatrie

President: Prof. Dr. A. Hottinger, Petersplatz 12, Basel.
Secretary: Dr. F. Hauser, Kinderspital, Basel.

Scottish Paediatric Society

President: Dr. Charles McNeil.
Secretary: Dr. J. H. Hutchinson, Royal Hospital for Sick Children, Glasgow, C.3.
Place: Edinburgh, Glasgow, Aberdeen, or Dundee.
Time: Twice a year.

Sociedad Boliviana de Pediatria

President: Dr. Carlos Ferrufino, Casilla 272, La Paz, Bolivia.
Secretary: Dr. Cecilio Abela D., Casilla 272, La Paz, Bolivia.
Place: S. C. I. S. P. Bldg. Time: Every Thursday.

Sociedad Chihuahuense de Pediatria

President: Dr. Carlos Villamar Talledo.
Vice President: Dr. H. Uranga.
Secretary: Dr. Reynaldo Ramos Varela.
Treasurer: Dr. David Russek.
Place: Children's Hospital, Matamoros St., Chihuahua, Chihuahua, Mexico.

Sociedad Chilena de Pediatria

Secretary-General: Dr. Oscar Undurraga A., Merced 565, Santiago, Chile.
Place: Medical Society, Merced 565, Santiago, Chile. Time: 7 p. m., every Thursday.

Sociedad Cubana de Pediatria

President: Dr. Miguel Fabres Izaga.
President: Prof. Agustin Castellanos.
Secretary: Dr. Carlos Hernandez Miyares, Hospital Nuestra Señora de las Mercedes L y 21. Apartado 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Nuestra Señora de las Mercedes y Hospital Municipal de Infancia, Habana. Time: 9 p. m., last Wednesday of every month.

Sociedad Dominicana de Pediatria

President: Dr. Jaime Jorge, Jose Joaquin Perez, no. 25, Ciudad Trujillo, R.D.
Secretary-Treasurer: Dr. Alberto Peguero, V., Ave. Bolivar 113, P. O. Box 464, Ciudad Trujillo, R.D.
Place: Children's Ramfis Hospital. Time: 8 p. m., last Friday of every month.

Sociedad Ecuatoriana de Pediatria

President: Dr. Alfredo Ceballos Carrión.
Secretary: Dr. Rafael Compte Andrade, P. O. Box 3480, Guayaquil.
Place: Hospital Central. Time: 11:30 a. m., every Friday.

Sociedad Mexicana de Pediatria

President: Dr. Gabriel Araujo Valdivia.
Vice-President: Dr. Ramón Pous Roca.
Secretary: Dr. Luis Torregrosa F.
Corresponding Secretary: Dr. Antonio Prado Vertiz.
Treasurer: Dr. Fernando López Clares.
Place: Hospital Infantil. Time: Last Thursday of every month.

Sociedad de Pediatría de Concepción (Chile)

President: Dr. Raúl Ortega A., Hospital Clínico Regional.
Secretary: Dr. Fidel Urrutia, Hospital Clínico Regional.
Place: Hospital Clínico Regional, Concepción. Time: 7:30 p. m., second Tuesday of every month from April to December, inclusive.

Sociedad de Pediatría de Córdoba

President: Dr. Juan Francisco Herrera, Córdoba, Argentina.
Secretary: Dr. Albino F. Dottori, Dean Fumes 2000, Esq. Ocaña (N.).
Place: Comite pro Defenso del Niño, Dean Fumes 2000. Time: Once a month.

Sociedad de Pediatria del Litoral

President: Dr. Jose E. Celoria, Hospital Centenario, Rosario, Argentina.
Secretary: Dr. Gloria Lovell, Hospital Centenario, Rosario, Argentina.

Sociedad de Pediatría de Santiago de Cuba

President: Dr. Antonio Béguez César, Sagarra 107.
Secretary: Dra. Susana Texdó Vaillant, Padre Pico 57.
Place: Colegio Médico. Time: Last Tuesday of every month.

SOCIEDADE PORTUGUÊSA DE PEDIATRIA

President: Prof. Castro Freire.
Secretary-Generale: Dr. Cordeiro Ferreira, Hospital de D. Estefania, Lisbon, Portugal.

SOCIEDAD URUGUAYA DE PEDIATRIA

President: Dr. Conrado Pelfort.
Secretary: Dr. José M. Portillo.
Place: Club Medico del Uruguay, Avenida Agraciada 1464 (Piso 13), Montevideo.

SOCIEDAD VENEZOLANA DE PUERICULTURA Y PEDIATRIA

President: Dr. Guillermo Rangel.
Secretary: Dr. Guillermo Tovar, P. O. Box 5, Caracas.
Place: Hospital Municipal de Niños, Caracas. Time: Once a month.

SOCIEDAD YUCATECA DE PEDIATRIA

President: Dr. José Lavalle, Yucatan, Mexico.
Secretary: Dr. Francisco Solís, Yucatan, Mexico.
Place: Hospital del Niño, Yucatan, Mexico.

SOCIÉTÉ DE PÉDIATRIE DE MONTRÉAL

President: Dr. Paul Letondal, 370 E. Roy St., Montreal, Quebec, Canada.
Secretary-Treasurer: Dr. Edmond Baril, 301 Carré Saint-Louis, App. 1.
Time: First and Third Thursday of each month.

SOCIÉTÉ DE PEDIATRIE DE PARIS

President: Dr. G. Heuyer, 1 Ave. Emile Deschanel, Paris 7e, France.
Secretary: Dr. Maurice Lamy, 94 Rue de Varenne, Paris 7e, France.
Place: Hôpital des Enfants Maladies, 149 Rue de Sèvres. Time: 4:30 p. m., third Thursday of each month.

SOCIÉTÉ DE PÉDIATRIE DE QUÉBEC

President: Dr. Roland Thibaudeau, 203 des Erables, Quebec, Que., Canada.
Secretary-Treasurer: Dr. Euclide Déchêne, 312 du Roi.

SOCIÉTÉ POLONAISE DE PEDIATRIE

President: Dr. Jan Bogdanowicz, Dzialdowska 1, Warsaw.
Secretary: Dr. Zofia Leimbach, Mieroslawskiego 9 m.6, Warsaw.

SOUTH AFRICAN PAEDIATRIC ASSOCIATION

Chairman: Dr. Basil Melle, 89 Lister Bldg., 195 Jeppe St., Johannesburg, South Africa.
Secretary-Treasurer: Dr. Seymour Heymann, 17 Lister Bldg., 195 Jeppe St., Johannesburg, South Africa.

SWEDISH ASSOCIATION OF PEDIATRICS

President: Prof. Sture Siwe, Lund.
Secretary: Dr. K. A. Melin, Färjestadsvägen 16, Bromma.

SWEDISH MEDICAL SOCIETY, SECTION FOR PEDIATRICS AND SCHOOL HYGIENE

President: Docent H. Magnusson, Sachs' Hospital for Children, Stockholm.
Secretary: Dr. M. d'Avignon, Norrtulls Sjukhus, Stockholm.
Place: Swedish Medical Society, Klara Östra Kyrkogata 10, Stockholm and Pediatric Clinic Uppsala. Time: Second Friday of each month (except summer).

THIRD SOUTH AMERICAN CONGRESS OF PEDIATRICS

President: Dr. Conrado Pelfort, Montevideo, Uruguay.

VIENNA PEDIATRIC SOCIETY

President: Dr. August Reuss, Universitäts-Kinderklinik, Lazarettgasse 14, Vienna 9.
Secretary: Dr. Erwin Martischnig, Universitäts-Kinderklinik, Lazarettgasse 14, Vienna 9.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON PEDIATRICS

Chairman: Dr. Woodruff L. Crawford, 321 W. State St., Rockford, Ill.
Secretary: Dr. Margaret Mary Nicholson, 1801 I St. N. W., Washington, D. C.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Warren W. Quillian, 140 Alhambra Circle, Coral Gables (Miami) 34, Fla.
Secretary: Dr. E. H. Christopherson, 610 Church St., Evanston, Ill.

AMERICAN HOSPITAL ASSOCIATION, MASSACHUSETTS HOSPITAL ASSOCIATION

Chairman: Dr. Frank Wing, Boston.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly 79, Mass.

AMERICAN PEDIATRIC SOCIETY

President: Dr. Hugh McCulloch, La Rabida Jackson Park Sanitarium, Chicago.
Secretary-Treasurer: Dr. Aims C. McGuinness, 237 Medical Laboratories, University of Pennsylvania, Philadelphia.
Place: Hotel Chamberlin, Old Point Comfort, Va. Time: May 7, 8 and 9, 1952.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. P. E. Williams, 193 James St. S., Hamilton, Ontario.
Secretary-Treasurer: Dr. R. L. Denton, 1615 Cedar Ave., Montreal.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Horace Hodes, New York.
Secretary-Treasurer: Dr. Sydney S. Gellis, 300 Longwood Ave., Boston 15.

SECTIONAL

INTERMOUNTAIN PEDIATRIC SOCIETY

President: Dr. Spencer Snow, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. Esther Gross, 202 E. South Temple St., Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. Henry E. Gallup.
Secretary-Treasurer: Dr. Harry Shwachman, 300 Longwood Ave., Boston.
Time: Four meetings a year.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. Carl Ashley, 2455 N.W. Marshall St., Portland, Ore.
Secretary-Treasurer: Dr. Gorham Babson, 1107 S.W. 13th Ave., Portland, Ore.
Time: Two meetings a year, in March and September.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. J. Harry Murphy, 915 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. Elizabeth Lowry, 301 Kenwood Pkwy., Minneapolis.
Place: Minneapolis, St. Paul, Duluth, and Rochester. Time: January, April, July, and October.

ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. Joseph Lyday, 227 16th St., Denver 2.
Secretary-Treasurer: Dr. Edwin T. Williams, 1850 Gilpin St., Denver 6.
Place: Children's Hospital of Denver or the Colorado General Hospital. Time: Monthly from October to June.

SOUTHERN MEDICAL ASSOCIATION, SECTION OF PEDIATRICS

Chairman: Dr. Carroll M. Pounders, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. Samuel F. Ravenel, 371 N. Elm St., Greensboro, N. C.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Chester I. Mead, 1930 Truxton Ave., Bakersfield, Calif.
Secretary: Dr. Clement H. Molony, 416 N. Bedford Dr., Beverly Hills, Calif.
Place: University Club of Los Angeles. Time: 6:30 p. m., second Wednesday in January, March, May, September, and November.

STATE

Alabama Pediatric Society

President: Dr. David B. Monsky, 748 Adams St., Montgomery.
Secretary-Treasurer: Dr. Alice Hill Pye, 305 Church St., Montgomery.
Place: Montgomery.

Arizona Pediatric Society

President: Dr. Carl A. Holmes, 1401 N. Seventh Ave., Phoenix.
Secretary-Treasurer: Jacob M. Sobol, 607 Heard Bldg., Phoenix.
Place: Phoenix or Tucson. Time: On Call.

Arkansas State Pediatric Association

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

California State Medical Society, Section on Pediatrics

Chairman: Dr. Alice Potter, 1418 Ninth Ave., San Francisco 22.
Secretary: Dr. Carl Erickson, 595 E. Colorado, Pasadena.

Florida State Pediatric Society

President: Dr. Edgar W. Stephens, 910 Harvey Bldg., West Palm Beach.
Secretary: Dr. Hugh A. Carithers, 2000 Park St., Jacksonville 4.
Place: Concurrent with state association convention and fall meeting.

Georgia Pediatric Society

President: Dr. Howard J. Morrison, 444 Drayton St., Savannah.
Secretary-Treasurer: Dr. J. Harry Lange, 490 Peachtree St. N. E., Atlanta.
Place: Atlanta. Time: At time of annual state meeting.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. H. L. F. Locke, 179 Allyn St., Hartford.
Secretary: Dr. L. W. Minor, 119 Main St., Middletown.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. Eugene T. McEnery, 4458 W. Madison St., Chicago 24.
Secretary: Dr. George L. Drennen, Jacksonville.

Indiana State Pediatric Society

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Matthew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Charlotte Fisk, 1201 Equitable Bldg., Des Moines 9.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport

Louisiana State Pediatric Society

President: Dr. William C. Rivenbank, 1448 Louisiana Ave., New Orleans 15.
Secretary-Treasurer: Dr. Bertha Wexler, 3439 Prytania St., New Orleans 15.

Medical Society of the State of New York, Pediatric Society

Chairman: Dr. Thurman B. Givan, 115 Remsen St., Brooklyn 2.
Secretary: Dr. Reginald A. Higgons, Port Chester.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. Waldo E. Nelson, Temple University, Philadelphia 40.
Secretary: Dr. Carl C. Fischer, 100 W. Coulter St., Philadelphia 44.

Michigan State Medical Society, Pediatric Section

President: Dr. Horace L. French, 301 Seymour Ave., Lansing.
Secretary: Dr. Park S. Bradshaw, 1014 Jefferson St., Muskegon.
Place: Detroit. Time: September, 1952.

Mississippi State Pediatric Society

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

Montana Pediatric Society

President: Dr. Edward A. Hagmann, 208 N. Broadway, Billings.
Secretary: Dr. George W. Nelson, 2814 Ninth Ave. N., Billings.
Place: Decided at each meeting—Spring and Fall.

Nebraska Pediatric Society

President: Dr. George J. Klok.
Secretary-Treasurer: Dr. Donald C. Nilsson, 115 N. 40th St., Omaha.
Place: Children's Memorial Hospital. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

New Hampshire Pediatric Society

President: Dr. Douglass W. Walker, Laconia.
Secretary-Treasurer: Dr. William D. Penhale, Concord.

New Jersey Medical Society, Pediatric Section

Chairman: Dr. Israel J. Wolf, 231 E. 31st St., Paterson.
Secretary: Dr. N. T. Crane, 147 E. 7th St., Plainfield.

New Mexico State Pediatric Society

President: Dr. M. K. Wylder, 221 W. Central Ave., Albuquerque.
Secretary-Treasurer: Dr. Ly Werner, 221 W. Central Ave., Albuquerque.

North Carolina Pediatric Society

President: Dr. Rowena Sidbury Hall, 15 N. Fifth St., Wilmington.
Secretary-Treasurer: Dr. Katherine Anderson, 138 Hawthorne Rd., Winston-Salem.

Northern California Pediatric Society

President: Dr. William C. Deamer, 3 Fifth Ave., San Francisco 18.
Secretary: Dr. A. L. Gleason, 1066 Longridge Rd., Oakland 10.
Place: San Francisco. Time: Second Thursday of February, April, September, and November.

North Dakota Pediatric Society

President: Dr. L. G. Pray, Fargo Clinic, Fargo.
Vice President: Dr. B. A. Mazur, Dakota Clinic, Fargo.
Secretary-Treasurer: Dr. William J. Ball, Northwest Clinic, Minot.
Place: Bismarck.

Oklahoma State Pediatric Society

President: Dr. J. B. Snow, 625 N. W. 10th St., Oklahoma City.
Secretary: Dr. Bertha Levy, 1200 N. Walker St., Oklahoma City.
Place: With Oklahoma State Medical Association. Time: Annual Meeting.

Philippine Pediatric Society

President: Dr. Alberto V. Tupas, Philippine General Hospital, Manila.
Secretary: Dr. Artemio P. Jongco, Philippine General Hospital, Manila.
Place: Philippine General Hospital. Time: Every two months.

South Carolina Pediatric Society

President: Dr. B. Owen Ravenel, 95 Rutledge Ave., Charleston 16.
Secretary-Treasurer: Dr. John R. Harvin, 206 N. Sumter St., Sumter.

Tennessee Pediatric Society

President: Dr. W. O. Vaughan, 2103 Hayes St., Nashville 5.
Secretary-Treasurer: Dr. C. Barton Etter, 1678 Peach, Memphis 12.
Place: Chattanooga. Time: Second Monday in April.

Texas Pediatric Society

President: Dr. C. S. E. Touzel, 1111 Pennsylvania Ave., Fort Worth.
Secretary: Dr. M. C. Carlisle, 1410 Austin Ave., Waco.
Treasurer: Dr. Edith Bonnet, 228 Medical Arts Bldg., San Antonio.

Virginia Pediatric Society

President: Dr. E. Berkeley Neal.
Vice President: Dr. E. L. Kendig.
Secretary-Treasurer: Dr. W. A. Mitchell.
Executive Board: Dr. F. R. Hopkins and Dr. J. O. Rydeen.

Wisconsin State Medical Society, Section on Pediatrics

President: Dr. Franklin J. Mellencamp, 1513 E. Capitol Dr., Milwaukee.
Secretary: Dr. E. H. Pawsat, 44 S. Marr St., Fond du Lac.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. Charles H. York, 10465 Carnegie Ave., Cleveland 6.
Secretary: Dr. Earl E. Smith, 10640 Carnegie Ave., Cleveland 6.
Place: Allen Memorial Medical Library. Time: 8:15 p. m., October, December, February, and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. A. L. Chute, 170 St. George St., Toronto, Canada.
Secretary: Dr. John Fletcher, 262 St. Clair St., W.
Place: Morrisania City Hospital. Time: 8:30 p. m., second Wednesday of each month,

Baltimore City Medical Society, Pediatric Section

President: Dr. Wilson Grubb, 4 E. 33rd St., Baltimore 18.
Secretary: Dr. Gibson J. Wells, 3101 St. Paul St., Baltimore 18.

Bronx Pediatric Society

President: Dr. Nathan M. Greenstein, 1488 Metropolitan Ave., New York.
Secretary: Dr. George Weiss, 790 Grand Concourse, New York 51.
Place: Morrisania City Hospital. Time: 8:30 p. m., second Wednesday of each month, except June, July, August, and September.

Brooklyn Academy of Pediatrics

President: Dr. Bernard Benjamin, 845 Park Place.
Secretary: Dr. Henry Cohen, 1761 E. 19th St. (29).
Place: Granada Hotel. Time: 8:45 p. m., fourth Wednesday of February, March, April, October, and November.

Buffalo Pediatric Society

President: Dr. Douglas P. Arnold, 159 Bidwell Pkwy.
Secretary: Dr. Richard A. Downey, 786 Forest Ave.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

Central New York Pediatric Club

President: Dr. Frank Williams, 58 Swan Ave., Albany.
Secretary-Treasurer: Dr. Fred W. Bush, 26 S. Goodman St., Rochester.
Place: Various cities in New York. Time: Third Tuesday of April and October.

Central Ohio Pediatric Society

President: Dr. E. V. Turner, 2479 E. Main St., Columbus.
Secretary-Treasurer: Dr. Mary L. Scholl, 1384 Grandview Ave., Columbus.
Place: As announced. Time: Third Wednesday of September, November, January, March, and May.

Chicago Pediatric Society

President: Dr. Alvah L. Newcomb, 723 Elm St., Winnetka.
Secretary: Dr. Alfred S. Traisman, 6349 N. Clark St.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May inclusive.

Cincinnati Pediatric Society

President: Dr. John H. Hunt, Kemper Lane Bldg.
Secretary: Dr. William F. Hunting, 6048 Montgomery.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Dade County Pediatric Society

President: Dr. William W. McKibben, 641 N. Greenway Dr., Coral Gables, Fla.
Secretary-Treasurer: Dr. Wesley S. Nock, 671 N.E. 57th St., Miami 38, Fla.
Time and Place: Variable.

Dallas Pediatric Society

President: Dr. Floyd A. Norman, 4325 Cole Ave.
Secretary: Dr. Dan S. Sanders Jr., 2103 Hayes St.
Place: Children's Hospital of Texas. Time: 6:30 p. m., the first Thursday of each month.

Detroit Pediatric Society

President: Dr. Wilfred D. H. Nolting, 16840 E. Warren.
Secretary: Dr. Ruben Meyer, 938 Maccabees Bldg.
Place: Variable. Time: Five times annually.

Fort Worth Pediatric Society

President: Dr. Frank Cohen, 712 S. Henderson.
Secretary: Dr. H. H. Womack, 1422 Pennsylvania.
Place: Welch and West Restaurant. Time: Third Friday of each month.

Honolulu County Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu 14.
Secretary-Treasurer: Dr. John A. Peyton, Kauikeolani Children's Hospital, Honolulu.
Place: At doctors' homes in rotation. Third Thursday of each month, 6 p. m.

Houston Pediatric Society

President: Dr. Emanuel B. Brandes, 3101 Fannin St.
Secretary: Dr. Robert D. Lomas, 5101 Fannin St.
Place: River Oaks Country Club. Time: 7:30 p. m., fourth Tuesday of each month.

Kansas City (Missouri) Pediatric Society

President: Dr. Harry M. Gilkey, 1624 Professional Bldg.
Secretary: Dr. Leland Speer, 1022 Hoel Pkwy.
Place: Kansas City General Hospital. Time: On call.

Knoxville-Oak Ridge Pediatric Society

President: Dr. Henry S. Christian, 205 Medical Arts Bldg., Knoxville, Tenn.
Secretary: Dr. John Eggleston, Oak Ridge Hospital, Oak Ridge, Tenn.

Long Beach Pediatrics Society

President: Dr. Lynn B. Vaughan, 302 Cherry Ave.
Secretary-Treasurer: Dr. Sam S. Woolington, 541 E. Broadway.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles 26.
Secretary-Treasurer: Dr. Robert Simonds, 267 S. Beverly Dr., Beverly Hills, Calif.
Place: Los Angeles County Medical Association Bldg., 1025 Wilshire Blvd. Time: Second Monday of February, April, June, October, and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Irving S. Pearlman, 7602 Bay Parkway, Brooklyn.
Secretary: Dr. Irving Weinstock, 350 Ocean Ave., Brooklyn.
Place: Kings County Medical Society. Time: 8:45 p. m., fourth Monday of April, October, November, January, and March.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. John Washington, 2032 Belmont Rd. N. W.
Secretary-Treasurer: Dr. George Maksim, 1418 Good Hope Rd. S. E.
Place: Medical Society Bldg., 1718 M St. N. W. Time: Spring and fall.

Memphis Pediatric Society

President: Dr. C. Barton Etter, 1678 Peach, Memphis 12.
Secretary-Treasurer: Dr. Jacob Danciger, 188 S. Bellevue Blvd., Memphis 4.
Place: University Center, 4 S. Dunlap. Time: 7 p. m., second Tuesday of every month.

Milwaukee Pediatric Society

President: Dr. A. L. Kastner, 1810 West Wisconsin Ave., Milwaukee 3.
Secretary-Treasurer: Dr. A. L. Babbitz, 606 West Wisconsin Ave., Milwaukee 3.
Place: City Club of Milwaukee. Time: Second Wednesday in February, April, June, October, and December.

Mobile Pediatric Society

President: Dr. Daniel Sullivan, 109 S. Broad St.
Secretary: Dr. Robert O. Harris, III, 1514 Government St.

Nashville Pediatric Society

President: Dr. John M. Lee, Doctors Bldg., Nashville.
Secretary: Dr. Dan S. Sanders Jr., 2103 Hayes St.
Time: Third Thursday of every month.

New York Academy of Medicine, Section on Pediatrics

Chairman: Dr. Arthur F. Anderson, 122 E. 76th St.
Secretary: Dr. L. Emmett Holt Jr., 477 First Ave.
Place: New York Academy of Medicine, 2 E. 103rd St. Time: Second Thursday of each month from November to May, inclusive, 8:30 p. m.

Oklahoma City Pediatric Society

President: Dr. Bertha M. Levy, 717 N. W. 22d St., Oklahoma City 3.
Secretary: Dr. C. M. Bielstein, 301 N. W. 12th St., Oklahoma City 3.
Place: Oklahoma Club. Time: On call.

Philadelphia Pediatric Society

President: Dr. Irving J. Wolman, 1740 Bainbridge St., Philadelphia 46.
Secretary: Dr. F. H. Harvie, 133 S. 36th St., Philadelphia 4.
Place: College of Physicians, 19 S. 22nd St. Time: 8:30 p. m., second Tuesday from October to May, inclusive.

Pittsburgh Pediatric Society

President: Dr. J. A. Gilmartin, 3708-15th Ave.
Secretary-Treasurer: Dr. C. J. Stoecklein, Medical Arts Bldg., Pittsburgh 13.
Place: Municipal Hospital, Pittsburgh. Time: Second Friday in October, December, February, and April.

Queens Pediatric Society

President: Dr. Meyerson Coe, 217-02 91st Ave., Queens Village, N. Y.
Secretary: Dr. Moe Goldstein, 111-32, 76th Ave., Forest Hills, N. Y.
Place: The Seminole Club, 112th St. and 70th Rd., Forest Hills, N. Y. Time: Second Tuesday in October, December, February, and April.

Richmond Pediatric Society

President: Dr. Carolyn McCue, 1030 W. Franklin St., Richmond 20.
Secretary-Treasurer: Dr. Hubert T. Dougan, 1832 Monument Ave., Richmond 20.
Place: Medical College Hospital. Time: 8 p. m., third Thursday of each month, except June, July, and August.

Rochester Pediatric Society

President: Dr. Jerome Glaser, 85 San Gabriel Dr., Rochester 10, N. Y.
Secretary-Treasurer: Dr. Daniel B. Peeler, 1162 Lake Ave., Rochester 13, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Third Friday of each month from October to May.

St. Louis Pediatric Society

President: Dr. Donald L. Thurston, 500 S. Kingshighway, St. Louis 10.
Secretary-Treasurer: Dr. Leo F. Gowan, 337 W. Lockwood Ave., St. Louis 19.
Place: Candlelight House. Time: Second Thursday of each month from October to May, inclusive, 6:30 p. m.

San Antonio Pediatric Society

President: Dr. Lucius Hill, 328 Medical Arts Bldg., San Antonio 5.
Secretary-Treasurer: Dr. Edith M. Bonnet, 228 Medical Arts Bldg., San Antonio 5.
Place: Bexar County Medical Library. Time: 7:30 p. m., first Tuesday of every month.

Santa Barbara Pediatric Society

President: Dr. Helen Hart, City Health Department, de la Guerra Plaza.
Vice President: Dr. Clarence Hills, 22B W. Micheltorena St.
Secretary-Treasurer: Dr. L. C. N. Wayland, 1235 Chapala St.
Meetings: Four times annually.

Seattle Pediatric Society

President: Dr. Norman Clein, 1155 Tenth St., Seattle 2.
Secretary-Treasurer: Dr. John A. Sterner, 509 Olive Way, Seattle 1.
Place: College Club. Time: Fourth Friday of each month from September to June at 6:30 p. m.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Fred J. Chapin, 101 John St., Bay City.
Secretary-Treasurer: Dr. George H. Lowrey, University Hospital, Ann Arbor.

Waco Pediatric Society

President: Dr. A. O. Manske, 1917 Austin Ave.
Secretary: Dr. M. C. Carlisle, 1410 Austin Ave.
Place: 1917 Austin Ave. Time: Third Tuesday of each month.

Westchester County Medical Society, Pediatrics Section (New York)

President: Dr. Harry W. Kaessler, 147 Prospect Ave., Mount Vernon, N. Y.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: The Roger Smith Hotel, White Plains, N. Y. Time: Fourth Thursday of every other month from September to June (dinner meetings).

Western Michigan Pediatric Society

President: Dr. Horace L. French, 301 Seymour Ave., Lansing.
Secretary-Treasurer: Dr. John C. Montgomery, 1810 Wealthy St. S. E., Grand Rapids.
Time: Meets on the first Wednesday in October, November, April, and May of each year. Two meetings are held in Grand Rapids and two in other designated localities.

Wichita Pediatric Society

President: Dr. Walter L. Schafer, 401 N. Emporia St., Wichita 2, Kan.
Secretary: Dr. William F. McGuire, 401 N. Emporia St., Wichita 2, Kan.
Place: Variable: Time: First Thursday of each month.

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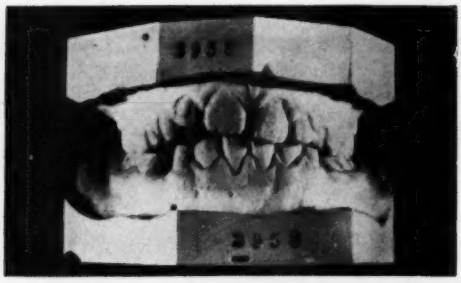

Nine year old boy had habit of holding index finger between teeth. Pressure caused teeth to portrude so that they do not meet.

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Child Health

Bad Habits in Good Babies. Herman M. Jahr. 16 pages. 15 cents.

What Does Your Baby Put in His Mouth? Chevalier Jackson and Chevalier L. Jackson. Tells how to prevent accidents from choking and what to do if they happen. 24 pages. 15 cents.

Keeping Your Baby Well. 22 pages. 15 cents.

The Case of the Crying Baby. Herman M. Jahr. 4 pages. 15 cents.

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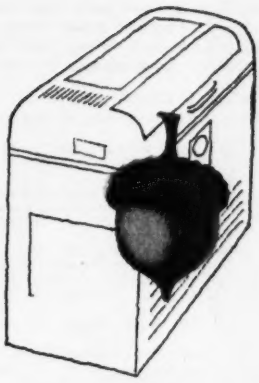

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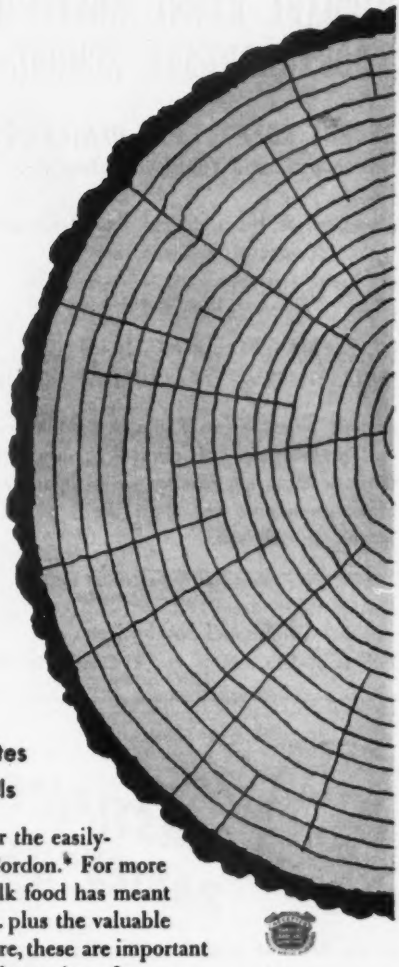

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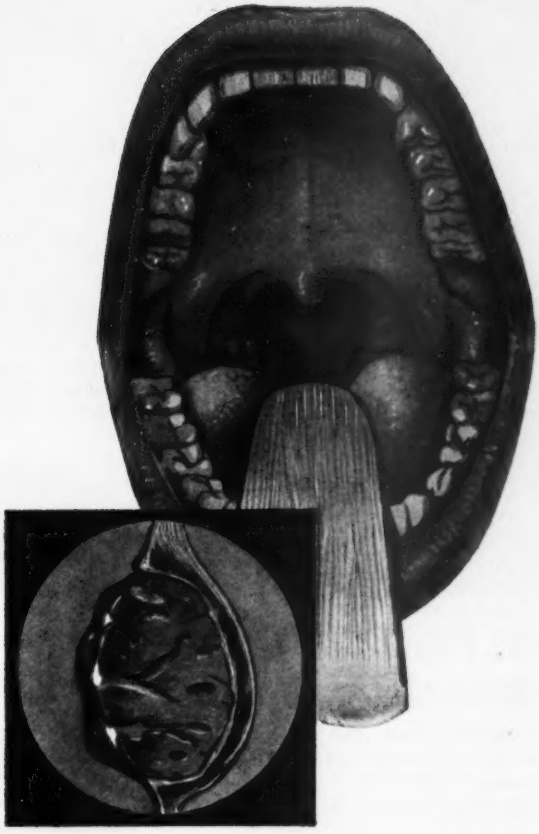

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Sayer, R. J.; Michel, J.; Moll, F. C., and Kirby, W. M. M.: Am. J. M. Sc. 221:256 (March) 1951

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1. Scheie, H. G., Tyner, G. S., Buesseler, J. A., and Alfano, J. E., *J. A. M. A. Arch. Ophth.* 45:301, March 1951.

2. Leopold, I. H., Purnell, J. E., Cannon, E. J., Steinmetz, C. G., and McDonald, P. R., *Am. J. Ophth.* 34:361, March 1951.

Literature on request

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In canned meat, about 60 to 70 per cent of the initial thiamine content, 90 to 100 per cent of the riboflavin and of the niacin, and 70 to 80 per cent of the pantothenic acid contents are retained.[1] With due allowance for the difference in the two cuts of beef compared, the following analyses of home-cooked beef round and of canned roast beef indicate their nutritional similarity.

Composition of 100 Gm., Edible Portion

	Kitchen-Cooked Beef Round[2]	Canned Roast Beef[2]
Water	59%	60%
Calories	233	224
Protein	27 Gm.	25 Gm.
Fat	13 Gm.	13 Gm.
Minerals, total	1.3 Gm.	2 Gm.
Phosphorus	224 mg.	116 mg.
Iron	3.4 mg.	2.4 mg.
Thiamine	0.08 mg.	0.02 mg.
Riboflavin	0.22 mg.	0.23 mg.
Niacin	5.5 mg.	4.2 mg.

The canning procedure has little effect upon the biologic value of the protein of meat.[3] In a recent study of the amounts of 18 amino acids in processed meats, canned beef, pork and lamb were generally similar in amino acid composition to fresh and kitchen-cooked samples.[4] Canned meat also is an excellent source of iron.

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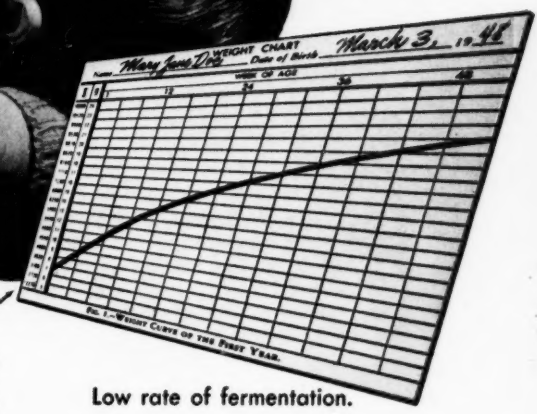

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